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THE
LARYNGOSCOPE.

VOL. XXXVIII SEPTEMBER, 1928. No. 9

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

PRIMARY JUGULAR BULB THROMBOSIS.*

DR. JACOB L. MAYBAUM and DR. IRVING B. GOLDMAN,
New York.

Because of an apparently unimportant, vague or indefinite aural history in cases of primary jugular bulb thrombosis, the condition is more likely to be seen at the onset by the internist or pediatrist rather than the otologist. For similar reasons, these cases are usually seen less frequently in special hospitals. The importance of early diagnosis becomes evident when we realize that in order to obtain a favorable outcome of this serious illness, prompt surgical interference is imperative.

The subject of primary jugular bulb thrombosis was first brought to the attention of the American medical profession by McKernon, in 1904. At that time he referred to the relationship which exists between an apparently unexplained septic temperature and an acute otitis media. The diagnosis, difficult at times even now, was all the more so at the time of McKernon's report, as his work antedated the very important blood culture studies of Libman, in 1906. Recent differential blood culture investigations of Ottenberg, to which more extended reference will be made later, have contributed still further to the early recognition of this condition.

The term primary bulb thrombosis is employed to designate the formation of an infected thrombus in the dome of the jugular bulb, the lateral and sigmoid sinus not being affected by the thrombosis in the initial stages of the lesion. It occurs usually in young children.

*Read before Section of Otology, New York Academy of Medicine, March 9, 1928.

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The bony partition separating the middle ear cavity from the jugular bulb is often extremely thin. An unusually high dome or roof of the bulb may encroach upon the floor of the middle ear. An added factor is the frequent presence of dehiscences in the floor of the tympanic cavity and as a result there may be direct contact between the floor of the middle ear and the dome of the bulb. The mastoid in young children not infrequently shows a congenital absence of cells. Under conditions such as these, it can be readily seen how an active purulent middle ear infection may primarily invade the blood current, attacking the bulb, the pathway of infection being through the small communicating veins and lymphatics or by a destructive process through the bone. An infection in the tympanic cavity may first involve the carotid plexus of veins situated along the anterior tympanic wall; from there the infection may pass directly into the bulb and thus produce a primary bulb thrombosis. It is important to remember that the jugular bulb may become directly infected as a result of an acute inflammatory process in the middle ear, without evidence at any time of middle ear discharge.

A study of the cases reported in this paper will serve the purpose of elucidating the clinical course pursued by this disease. The onset may be insidious, the aural history being so indefinite as to escape the attention of those who see these patients first. In one instance, Case A. H., there was a history two months previous to admission, of fleeting pain in the right ear of 24 hours' durations; at no time was there any middle ear discharge. A diagnosis of primary bulb thrombosis was finally made and the relationship between the prolonged sepsis and a negligible ear history was established and confirmed at operation. In the majority of instances, aural examination discloses evidence of a subacute otitis media purulenta or of a beginning middle ear resolution without mastoid involvement. The drums are flat, thickened and the short process or hammer handle is evident. The hearing may be only moderately impaired.

Aside from a history of an acute otitis media, the only symptom of importance is a fluctuating temperature between 99° F. or 100° F. and 104° F. Not infrequently the temperature runs a low course, 100° F. or 101° F. for a few days, followed by a rapid rise and fall. Most of our cases have come to us with such a history of two or three weeks' duration. As a rule these patients do not have a distinct chill; they may, however, have chillly sensations. If the rapid fluctuations in temperature continue for a few days, pallor becomes quite evident, and the tongue appears white and dry. The following symptoms associated with a more or less definite aural history arouses

ones' suspicion of the presence of a bulb thrombosis, even before the report of a postive blood culture: Pallor, dry tongue, an enlarged spleen, enlarged and tender lymph glands at the angle of the jaw and early changes in the ocular fundi. The blood count shows a leucocytosis, 12,000 to 15,000, and a high polynucleosis, 75 per cent to 85 per cent. Daily estimation of hemoglobin percentage and red blood cell count points to a progressive secondary anemia. The diagnosis can be made, in the presence of a sepsis unaccounted for by any other demonstrable lesion, by a history of an acute middle ear suppuration and a positive blood culture.

Valuable aid in the diagnosis and localization of sinus thrombosis has resulted from the recent blood culture studies of Ottenberg. Blood is taken simultaneously from both internal jugular veins, cultured and the colonies counted. In a suspected case of sinus thrombosis, a preponderance of bacteria may be found in the blood obtained from the vein on the normal side as compared with that from the diseased side. The procedure was done by Ottenberg in three of our cases of suspected primary bulb thrombosis with striking findings. These findings apparently point first, to the presence of an occluding thrombus; secondly, in bilateral aural involvement, the disproportionate bacterial growth is strongly suggestive, from the cases thus far studied, of an involvement of the sinus on the side from which the lesser number of colonies is obtained. This work further indicates the extreme importance of careful surgical procedure to arrest systemic invasion, by thoroughly dealing with the torcular end of the diseased sinus, the jugular vein having been previously ligated.

From Ottenberg's studies it appears that in the presence of an occluding thrombus involving the jugular bulb and lateral sinus, the general systemic invasion is produced, in large measure, as a result of the free circulation of blood at the torcular end of the thrombus; the infection is carried by way of the sinuses on the normal side; the circulation at the upper end of the juglar vein and bulb on the diseased side is far less active.

To be effective, surgical efforts must be instituted early. Briefly, our procedure is as follows: The diagnosis having been made, a low ligation of the internal jugular vein is done through a horizontal incision. If doubt exists as to the diagnosis, the mastoid should be first exenterated, sigmoid sinus uncovered and inspected. In eight of our nine cases the diagnosis was definitely determined before operation. The vein having been ligated, the mastoid is rapidly exenterated, the sinus is exposed and followed back until it appears to be normal. The sinus is then incised after plugs have been prop-

erly placed at the torcular and bulb ends. If the condition of the patient permits, the sigmoid groove is followed to the bulb; the latter is then curetted and drained. It is of utmost importance to obtain free bleeding from the torcular end of the exposed sinus.

As to the indications for a jugular bulb operation and the surgical approach to the bulb: In a majority of instances operative procedures upon the bulb are unnecessary. If sepsis continues despite thorough surgical measures directed to the torcular end of the sinus and evidence points to suppuration in the jugular bulb, our indications are to deal with the focus in the bulb. The question of the presence of suppuration in the bulb is determined by our ability to cause pus to well up into the mastoid cavity from the bulb, upon pressure over the tender swelling which is present at the angle of the jaw.

The formidable surgical procedures advised and described by Voss, Grunert and Tandler have not been found necessary in our experience. Besides being extensive in character they entail a certain amount of danger to the facial nerve. Ordinarily we expose the sigmoid sinus to the posterior bony canal wall and then curette the remainder of the surgical sinus and as much of the bulb as we can reach. Nothing more was done in all but two of the cases. In these two cases, the procedure of doing a complete sigmoid operation, suggested by Friesner a few years ago, answered the purpose. This method of exploring the bulb is based upon the early steps in the operations of Voss and Grunert. Because of its comparative simplicity and the rapidity with which it can be performed, together with the entire freedom from danger of injury to the facial nerve, this modification recommends itself as an ideal procedure for exploring the bulb. It differs from those previously referred to in that it is less radical and yet thoroughly efficacious in draining the bulb. Where the sigmoid sinus approaches the posterior canal wall at a more acute angle than usual, the procedure may not be feasible.

Essentially the method of approach to the bulb is as follows: Having made a transverse incision through the soft parts of the posterior flap, the splenius capitis muscle is detached from the posterior border of the mastoid; the underlying periosteum is then elevated for a short distance from the base of the skull toward the bulb (this elevation is done as freely as in a submucous resection). With the heavy rongeur the posteroexternal wall of the sigmoid groove is followed down, thus exposing the dura of the posterior fossa. At the lower termination of the sigmoid sinus before it enters the bulb the posteroexternal wall of the sinus becomes its inferior wall. The latter, together with the jugular process of the occiput, is removed

with a few more bites of the rongeur. Even before the last step, one can readily see that the facial nerve is at a safe distance from the field of operation, being considerably external and above. The bulb now readily presents into view, and can be thoroughly curetted and drained. In reality the above procedure should be called a complete sigmoid sinus operation. As ordinarily performed, the diseased sigmoid sinus for a distance of 1 inch or more from the bulb, may contain a thrombus in contact with cerebellar dura and is left untouched. It is not necessary, as in more extensive operations, to join the postauricular incision with the incision in the neck. The technique outlined should not require more than five minutes.

These young patients cannot stand prolonged operative procedure: the various steps in the operation outlined, therefore, must be done as expeditiously as possible. If necessary an immediate intravenous saline during the course of the operation may be employed. The use of a continuous intravenous saline during the operation has been suggested. This measure would probaly lessen the likelihood of collapse occurring during the course of or immediately following operation.

Blood transfusion is not necessarily indicated in all cases of sepsis of otitic origin. These patients should not be subjected to the added danger of transfusion, even though the possibility of an untoward result might be remote, when the clinical course and physical condition following well directed surgical management give every indication of being satisfactory. Blood transfusion may be useful preoperatively. Patients in serious condition as a result of sepsis of comparative long duration withstand operations better if given a transfusion preoperatively. A blood transfusion is usually done immediately after operation. As to subsequent transfusions, in recent years we have recourse to these only if the general condition, especially as indicated by a decreasing hemoglobin and red blood cell count warrant it. We do not favor the indiscriminate use of blood transfusions in these cases.

The following cases of primary jugular bulb thrombosis are reported from the service of Dr. I. Friesner at the Mt. Sinai Hospital. The outline gives in comprehensive form the essential facts regarding them.

Eunice M., age 9 years; admitted Aug. 4, 1927; discharged Sept. 3, 1927. Patient was admitted to the Pediatric Service of Mt. Sinai Hospital because of chills and fever. At the age of 3 years she had had pneumonia, which was complicated by a bilateral otitis media. The right ear had discharged for several days during the preceding year. Following an acute upper respiratory infection, the right ear

began to discharge two weeks ago. Since that time, the temperature has been elevated to 106°, and for the past week the child has had from four to five chills daily.

The patient appeared well nourished but acutely ill. The general physical examination, except for a tachycardia and elevated red spots on the buttocks and forearm, was negative. The left ear was normal. The right ear disclosed a moderate amount of mucopurulent discharge from a large anterior perforation; the landmarks could be seen; the hearing was only moderately impaired. There was no postauricular edema or mastoid tenderness. Peripheral and jugular blood cultures were taken and reported positive for hemolytic streptococcus, the peripheral showed 16 colonies per c.c., the right internal jugular, 15 colonies, and the left internal jugular, 67 colonies.

In view of the definite ear history, the absence of postaricular signs, the chills, fever and bacteremia, a diagnosis of primary jugular bulb thrombosis was made. At operation the mastoid was found to be sclerosed, the sinus plate was intact, the sinus wall was grayish-white and lusterless, and contained an obturating thrombus. There was no bleeding from the bulb, but free bleeding was obtained from the torcular end. In spite of the fever and bacteremia, the child's condition continued to improve and further surgical interference was not attempted. After 12 days of fluctuating temperature, the fever subsided and the child was discharged cured.

Arnold G., age 6½ years; admitted Feb. 27, 1926; ceased. This child had been in the hospital from Jan. 28 until Feb. 17, 1926, because of bilateral purulent otitis media following measles. After being in the hospital several days, the right ear resolved, the temperature dropped, the general condition improved and the boy was discharged. His left ear had discharged when he was 6 months old, and again for about four months when he was 5 years old. Three days prior to admission, the otorrhea of the left ear, which had been rather profuse, ceased, the temperature rose to 106° and the boy began to complain of frontal headache.

Physical examination showed an undernourished, acutely ill child. The right ear had a large perforation in the inferior quadrant without any discharge. The left ear showed a moderate thin, purulent discharge, from a large central perforation inferiorly. The drum was flat, the landmarks were visible, the canal normal. There was no tenderness or thickening over the mastoid. The hearing was slightly impaired in both ears. There was a spontaneous horizontal nystagmus of equal amplitude to the right and left in extreme positions of the eye. The liver was enlarged, palpable glands were present in both sides of the neck, larger and more numerous on the right.

Except for slight fullness of the retainal veins, the fundi were negative.

On the following day the boy had a chill and a rise in temperature to 104.6°. The temperature fluctuated for 48 hours, remained low for the next six days, and then rose again to 103.4°. Because of the continuing severe frontal headaches, the onset of dizziness and vomiting, and the absence of any other focus, it was considered advisable to explore the lateral sinus, despite the absence of positive blood culture. This was done on March 11.

At operation the mastoid was found sclerosed, and the left sinus wall appeared grayish-blue. The right sinus was also explored and the sinus wall appeared normal. The left sinus was then aspirated but no blood was obtained; the sinus wall was slit and an obturating thrombus was found. After plugging the torcular and jugular ends, the thrombus was removed. Removal of the plug from the jugular end gave no bleeding. The bulb was then curetted. Owing to the poor condition of the child, the plug at the torcular end was not removed.

Following the operation, the child became worse. He was irritable, vomited and complained again of headache. Neurological examination showed a very mild hemiparesis on the right side, a right facial paresis, diminished right abdominal reflexes, and a definite right Babinski. Lumbar puncture showed the spinal fluid to be under increased pressure. Cell count showed eight cells—all lymphocytes. The cerebellum was aspirated; no abscess was encountered. Blood culture taken at this time was positive for hemolytic streptococci.

The symptoms became worse. The signs continued. There was a progressive increase in the fundi, papillary edema now being present. The pulse rate dropped from 112 to 64. On April 21, the left temporosphenoidal lobe was explored. At a depth of 1½ inches from the dura, an abscess containing about 75 c.c. of thick yellow pus was found; this was drained. The child continued to do poorly and ceased.

Portia S., age 9 years; admitted March 26, 1927; discharged April 22, 1927. Patient was admitted to the Pediatric Service of the Mt. Sinai Hospital because of high fever of four weeks' duration. The past history was negative except for pain and discharge of the left ear at 4 years of age. The child was well until five weeks before admission, when she complained of pain in her left ear and the drum perforated spontaneously. After the first week, there was no further pain in the ear, and the discharge stopped, but she began to have daily fluctuation of temperature, ranging from 102° to 104°. There had been chilly sensations but no real chills. In the past two

weeks she had complained of frequent joint pains, particularly in the right ankle, and she had had a swelling of the left elbow joint, which lasted for one day.

The child appeared acutely ill, anxious, but quite alert. The right ear was normal. The left tympanum was flat, thickened, all landmarks were present. There was no mastoid tenderness. The hearing was good. The middle ear showed evidence of a recent inflammatory process. Fundus examination showed a beginning optic neuritis. On the dorsum of the legs and on the right thigh there were three pustules, 1 c.m. in diameter. The blood culture taken on admission showed hemolytic streptococcus, 3 colonies per c.c. In view of the positive blood culture with the definite ear history and beginning fundus changes, primary jugular bulb thrombosis was diagnosed.

On March 27, the left lateral sinus and jugular bulb were explored and the internal jugular vein was ligated. The mastoid was found to be sclerosed, the sinus plate was intact, and when the sinus was uncovered frank pus exuded. Following the operation, the child was transfused. The temperature subsided rapidly and the child was discharged after an uneventful recovery.

A. S., age 7 years; admitted Feb. 4, 1926; discharged April 7, 1926. Patient was admitted to the Pediatric Service of the Mt. Sinai Hospital, with a history that three weeks prior to admission he began to cough, and had slight fever. On the third day of his illness, he complained of pain in both ears. These began to discharge at the end of four days. The temperature at that time was elevated to 104-105°, which was followed by an afebrile interval of two or three days, only to be followed by another rise to 105°. This type of temperature continued until the time of admission. With each rise in temperature he complained of chilly sensations. About one week prior to admission, the ears ceased to discharge, and at about this time he began to experience vague pains around the umbilicus not associated with vomiting. Two days later he complained of pain in the left hip, which persisted up to the time of admission.

He had had attacks of acute otitis media at the age of 2 and 3 years.

The physical examination showed an undernourished, undeveloped boy of 7 years, with a waxy pallor resembling very closely the pallor seen in leukemia. He did not appear acutely ill. The cervical, axillary and inguinal glands were somewhat enlarged but only the cervical glands on the left side were slightly tender. The liver and spleen were enlarged about 5-6 c.m. below the costal margins, the edges being hard, firm and sharp, but not tender. The left hip joint was negative except for moderate pain on adduction of the leg.

				SYMPTOMS	
NAME	AGE	SEX	PAST OTITIS	LOCAL	GENERAL
E. M.	9 yrs.	F	+	2 weeks painless discharging ear.	Chills and fever.
A. G.	6½ yrs.	M	+	(a) 6 weeks discharging ear, which stopped 3 days before admission. (b) Pain in left ear.	Headache vomiting fever.
P. S.	9 yrs.	F	+	(a) Pain in ear 5 weeks. (b) Discharging ear for 1 week.	(a) Epistaxis. (b) Fleeting joint pains. (c) Daily excusion of Temp. 101-104. (d) Chilly. (e) Perspiration.
A. S.	7 yrs.	M	+	Discharging ear 2 weeks ago for 1 week.	(a) Pain in left hip for 1 week. (b) Abdominal pains 7 days ago for 5 days. (c) Chilly, Temp. 99-105 alternating with afebrile periods. (d) Occasional headache.
A. C.	17 yrs.	M	Deafness for 1 week 5 months previously.	Buzzing of right ear for 1 week. Discharging painless right ear for 3 days.	(a) Headache. (b) Drowsiness for 1 week. (c) Chills and high fever for 3 days. (d) Abdominal pains 3 days ago for 1 day.
E. G.	9 yrs.	F	+	Pain and discharging right ear for 10 days.	(a) Vomiting. (b) Septic temperature.
J. S.	6½ yrs.	M	—	Discharging ear for 2 weeks.	(a) Fever for 2 weeks. (b) Septic Temp. for 2 days. (c) Chills.
A. H.	7 yrs.	F	+	Otalgia for 2 days without discharge 11 days before admission.	(a) Fever 4 days. (b) Pain in both lower extremities. (c) Pain and swelling of ankle joint.
D. S.	21 yrs.	F	Chronic Otorrhea.	Pain in ear 1 week.	(a) Fever. (b) Chills. (c) Sweats. (d) Pain in right side of neck. (e) Frontal headache.

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SIGNS					
ERAL	LOCAL	GENERAL	METASTASES	FUNDUS	
ver.	Right ear. Flat drum, landmarks moderate, mucopurulent discharge.	Spleen — Liver — Glands — Cutaneous +		—	
	Left ear. Flat drum, landmarks visible, moderate purulent discharge from a central perforation.	Spleen — Liver + Glands + Cutaneous +		—	
joint pains xcusion of 4.	Flat drum. Landmarks visible. Hearing good.	Spleen — Liver + Glands — Cutaneous +		+	
on.					
ft hip for 1 al pains 7 5 days. mp. 99-10 ith afebrile	Perforation of right drum. No discharge from either ear. Evidence of recent acute otitis. Drum flat, and thickened.	Spleen + Liver + Glands — Cutaneous +		—	
l headache.					
s for 1 week. high fever al pains 3 l day.	Flat drum. Handle can be outlined. A very slight, thin purulent discharge from perforatic anterior quadrant. Hearing very much impaired. Resolving middle ear.	Spleen + Liver — Glands + Cutaneous —	Both lungs?	Beginning optic neuritis. Veins enlarged.	
mperature.	Flat drum. Thin pulsating discharge through posterior superior perforation. Hearing some what impaired.	Spleen — Liver — Glands — Cutaneous —		Early optic neuritis.	
2 weeks. mp. for 2	Right ear resolving. Left ear resolving.	Spleen — Liver — Glands + Cutaneous —		—	
lays. both lower swelling of	Flat, thickened drum. Landmarks visible. Hearing good.	Spleen + Liver + Glands + Cutaneous +	Pyarthrosis of left hip. Abscess of right ankle. Abscess of thumb.	6 weeks after admission, bilateral papilloedema. R 3 D; L 3 D.	
ght side of eadache.	Flat thickened drum. Perforation at umbo.	Spleen — Liver — Glands + Cutaneous —		Choked disc. O. D. 2 D. O. S. 4 D.	

NINE CASES OF PRIMARY BULB THROMBOSIS

	OPERATION						BLEE
Blood Culture	Mastoid	Vein	Sinus Plate	Sinus Wall	Thrombus	Bulb	
+	Sclerosed.	Small.	Intact.	Gray, lusterless.	Obturating.	No.	Fr
—	Normal.		Intact.	Grayish blue.	Obturating.	No.	
+	Cellular. Normal.	Collapsed.	Intact.	Reddish.	Obturating.	No.	Ye
+	Normal.		Intact.	Grayish, thickened.	Obturating.	No.	Bl su
+	Sclerosed.	Collapsed.	Intact.	Thick, yellow, perforated with thick pus, escaping.	Obturating, purulent.		B su ve co
+	Sclerosed.		Intact.	Normal, except for small grayish area. (sigmoid).	Not demonstrated.	Yes.	Ye
+	Sclerosed.		Intact.	Yellow pus.	Fluid pus.	Yes.	Ye
+	Sclerosed.	Collapsed.	Intact.	Thick, contained pus.	Obturating, purulent.	No.	No
+	Sclerosed.		Intact.	Discolored.	Purulent.	No.	Ye

SIS

BLEEDING			POST OPERATIVE COUR		
THROMBUS	BULB	TORCULAR	METASTASES AND COMPLICATIONS	FURTHER SINUS OPERATION	
Obturating.	No.	Free.	None.	None.	Neg
Obturating.	No.		Tempeoro-sphenoidal abscess.	None.	Pro peri
Obturating.	No.	Yes.	None.	None.	Adv loe
Obturating.	No.	Bleeding from superior petrosal.	None.	None.	Neg
Obturating, purulent.		Bleeding from superior petrosal vein necessary to discontinue operation.	(a) Arthritis of left shoulder. (b) Deep abscess of buttock. (c) Pulmonary metastases, bilateral, which resolved.	None.	Neg
Not demonstrated.	Yes.	Yes.	(a) Abscess over sacrum and thoracic spine. (b) Cervical arthritis.	Two.	Inc 2 di
Fluid pus.	Yes.	Yes.	None.	None.	Nor
Obturating, purulent.	No.	No.	Broken down cervical glands. Sympathetic meningitis for 3 days—2700 cells in spinal fluid, 65% polys.	None.	Inc regr
Purulent.	No.	Yes.	Abscess of thigh, forearm. Poniarthritis of ankle.	None.	Gra

OURSE

Fundi	Blood Cultures	Fever	Result
Negative.	Repeatedly positive.	Fluctuating fever for 12 days, then normal.	Well.
Progressive increase to peripapilloedema.	Positive on one occasion.	Fluctuating for 2 weeks, then around 100 for 5 weeks until pyrexia before death.	Ceased.
Advanced to peripapilloedema, then receded.	Negative.	Fluctuating temperature for 4 days, then dropped.	Well.
Negative.	Negative. One taken immediately after operation.	Dropped to normal immediately.	Well.
Negative.	Positive once.		Well.
Increase to elevation of 2 diopters, then recession.	Positive, finally negative.		Well.
None.	Persistently positive, finally negative.		Well.
Increased to 6 D, then regressed.	Positive 20 hours preoperative, then sterile.	Dropped immediately following operation.	Well.
Gradual recession.	Positive.	Temperature subsided immediately.	Well.

There was a large central perforation in the right drum but no discharge. The left ear was also dry. There was evidence, however, of a recent acute otitis media in both ears.

His blood count was of extreme interest and showed the following:

	Feb. 4	Feb. 6
HGB.	62%	
RBC.	3,520,000	
Polys.	48%	51%
Lymphs.	25%	20%
Monos.	5%	13%
Myelocytes	16%	13%
Myeloblasts	3%	2%
Myelogones	3%	2%

For five days after his admission he had a septic temperature. The fundi were repeatedly negative. A blood culture was reported positive for streptococcus hemolyticus, 60 colonies per c.c. A blood culture taken four days later was again reported positive for streptococcus hemolyticus, the number of colonies increased to 122 per c.c. In view of the streptococcemia with a definite increase in the number of colonies, the diagnosis of primary jugular bulb was strongly entertained. The question then arose as to which side was the site of the lesion. With this in view a culture was taken from the blood of both internal jugular veins by Dr. Ottenberg. It was found that the blood from the right internal jugular vein grew 275 colonies, and the left only 9 colonies. For this reason the right mastoid was first explored and the sinus wall exposed. There was no disease in the mastoid except for a slight sclerosis; the sinus wall appeared normal. A similar procedure was done on the left side and again the mastoid showed no disease, but here the sinus wall appeared gray and was definitely thickened. Aspiration of the sinus failed to show the presence of blood. The internal jugular vein on that side was then ligated and the sinus operation continued. A large obliterating thrombus extending from the jugular bulb to about the level of the knee was removed. The bulb was curetted and drained.

Blood culture taken immediately after the operation, and another 48 hours afterwards were both reported sterile. Except for the reaction following transfusion, the boy made an uneventful recovery; his temperature declined to normal and remained so throughout his stay in the hospital. His weight increased considerably; the wounds healed satisfactorily. He was discharged April 7, *i. e.*, seven weeks after the operation, very much improved.

Adolph C., age 15 years; admitted Jan. 19, 1924; discharged April 4, 1924. Patient was admitted to the Otological Service of the Mt. Sinai Hospital complaining of earache, drowsiness and buzzing in the right ear for one week. Three days before admission, there

was a spontaneous, painless perforation of the right drum, with moderate purulent discharge. The boy looked sick and had a temperature of 105.4°. There was a slight thin, purulent, nonpulsating discharge from an anterior perforation. The drum was flat, the landmarks visible, there was no sagging. The hearing was moderately impaired. The middle ear was evidently resolving. There were palpable glands at the angle of the right jaw. The blood count showed W. B. C., 22,000, with 88 per cent polys. The fundi were negative.

At 2:30 o'clock of the following afternoon, the patient had a chill and the temperature rose to 106.2°. It was then decided to explore the sinus because there was nothing else to account for the clinical course. A shower of subcrepitant rales appeared in both lungs anteriorly. The operation was deferred, inasmuch as it was thought that the patient had developed a pneumonia. The blood culture taken on admission was still negative. On the morning of the third day of the patient's admission to the hospital, it was noted that there was a beginning bilateral optic neuritis. The blood culture of the second day was reported positive for hemolytic streptococcus. The signs in the chest remained the same. The clinical picture was not one of pneumonia. On Jan. 22, three days after admission, the sinus was explored. The mastoid was found apparently free of pathology. The sinus plate was intact. When the sinus was uncovered pus was seen exuding from it. The jugular vein was ligated. The pus yielded an hemolytic streptococci on culture.

The temperature remained septic after the operation. The chest signs remained diffuse. Occasionally, the patient expectorated blood-tinged sputum. For the next four weeks the temperature remained high. The chest signs persisted. A metastatic arthritis of the left shoulder developed, which subsided. At the same time a large, deep abscess of the buttock was opened and drained. The blood culture was positive only once during the interval. The temperature gradually came down to normal. The chest signs disappeared. The general condition improved, and the patient was discharged as recovered.

Comment: The predominant feature of this case of primary jugular bulb thrombosis was the occurrence of a severe metastatic pulmonary involvement.

Esther G., age 12 years; admitted Oct. 3, 1925; discharged Dec. 31, 1925. This child was admitted to the Otological Service of Mt. Sinai Hospital because of pain and discharge from the right ear for ten days. The past history was negative except for discharging ears, which she had at 4 years of age. Ten days before admission, she had had an earache and a myringotomy had been performed. The right tympanum was flat; a thin pulsating discharge could be seen

through a posterior-superior perforation. The hearing was somewhat impaired. The fundi showed reddish nerve heads, with all disc margins blurred, the veins were very full, dark and dilated. The hemoglobin was 80 per cent. A blood culture taken on the following day revealed the presence of a hemolytic streptococcus.

On Oct. 5, the internal jugular vein was ligated. The mastoid was explored and found to be markedly sclerotic. The sinus wall was then exposed and showed evidence of a phlebitis. There was no demonstrable evidence of a thrombus. Full bleeding was noted from both ends.

Following the operation, the patient continued to run temperature, almost daily, up to 105°. The bacteremia persisted. Two explorations of the sinus were made but no thrombus was found. Three transfusions were given, which were followed by temporary improvement.

On Nov. 21, the child developed two metastatic abscesses, one on the sacrum and one over the thoracic spine. These were incised and drained. The fundi showed increase in elevation of 2 diopeters. It was not until Dec. 13 that the temperature finally dropped and the blood culture became sterile. Following this the child's condition improved markedly; the irritability, restlessness and complaints ceased, and the discs approached normal. The child was discharged completely cured.

Comment: It was difficult to determine, because of the excessive bleeding, whether we were dealing with a phlebitis alone or thrombosis of the bulb. On operation, the mastoid was found sclerosed. The area of phlebitis was definitely seen at the lower end of the sigmoid sinus. Free bleeding on excising the sinus necessitated a cessation of further operative interference. It was, therefore, impossible to determine whether the bleeding came from the petrosals, bulb or torcular end.

James S., age 6½ years; admitted May 25, 1921; discharged July 18, 1921. Patient was admitted to the Otological Service of Mt. Sinai Hospital complaining of discharging ear and fever of two weeks' duration. The ears had been incised two weeks before admission and were discharging moderately. The temperature, which had been continuous, had been septic for the past two days, and rises of temperature were accompanied by two distinct chills. On admission, the boy was acutely ill.

On examination of the right ear, it was found that the canal was normal, and that there was a mucoid discharge from a perforation posterior-inferiorly. There was no mastoid tenderness, the drum was flat, the short process presenting. The left ear disclosed a normal

canal, the discharge was more mucopurulent, there was a perforation posteroinferiorly. Mastoid tenderness and thickening were absent.

For the next ten days, in spite of the fact that the ear began to resolve, the temperature varied from 97° to 105°, the child complained of frontal headache, and the leucocytosis continued. Firm, tender glands developed, extending from the tip of the left mastoid down to the sternoclavicular junction. Several blood cultures were taken, the last of which showed hemolytic streptococcus in flasks only. Because of the bacteremia and the adenopathy along the course of the left internal jugular vein, the left mastoid was explored on June 5, and was found normal. The sinus was then uncovered and found full of fluid pus. The dissection was continued back to the torcular and the curette passed back into the torcular without encountering free bleeding. When the plug was removed from the bulb, it was filled with pus. On account of the child's condition the bulb was not explored.

The postoperative course was very stormy. Numerous Unger transfusions of small quantities of blood were given. The blood cultures were repeatedly positive even after the operation, one culture showing as many as 500 colonies per c.c. On account of the progressive improvement in the clinical picture, although the patient was still running a septic temperature and having a persistent bacteremia, no further exploration of the bulb was deemed advisable. There was a gradual improvement in the clinical condition, accompanied by a subsidence in the temperature, and on July 6 the blood culture was sterile.

Patient was discharged well.

Anna H., age 7 years; admitted Feb. 1, 1923; discharged Sept. 14, 1923. This patient was admitted to the Pediatric Service of Mt. Sinai Hospital with a history of severe pains in both legs for the past four days. Eleven days before admission, she had had pain in the right ear. The mother was assured by her physician that there was nothing abnormal with the ear. The pain continued for a few days and disappeared. There was no discharge. The temperature on admission was 103.2°. The physical examination revealed equisite pain on pressure over both hips, especially the left, and over the right tarsus. The right tympanum was somewhat thickened; landmarks were present. No evidence of mastoid disease was present. Blood culture taken at this time showed hemolytic streptococcus. The temperature remained between 102-104°, and on Feb. 5 a huge pyarthrosis of the left hip was opened and drained, hemolytic strep'ococci being recovered from this focus.

After this operation the temperature ranges were not so high for a few days, and then began again to oscillate between 101° and 103°, until on Feb. 11 a pyarthrosis of the right ankle was opened and drained, yielding the same organism. Whereas bacteremia was present persistently up to this point, the blood after this operation apparently became sterile for five weeks, during which time there was marked improvement in the general condition, except that there developed marked lumbar tenderness, especially on the right side. The right kidney became enlarged and hyaline granular casts, W. B. C. and R. B. C. appeared in the urine in great numbers. Exploration of the right kidney for subcortical abscess was negative.

On March 19, the temperature suddenly rose to 104.8° and with this there appeared an adenitis at the angle of the right jaw. A blood culture revealed the appearance of hemolytic streptococcus. The temperature oscillated between 100-105° for a few days. The fundi showed bilateral choked discs, O. D. —3 D.; O. S. —6 D. The glands increased in size and the bacteremia became pronounced—100 colonies per c.c. In view of the bacteremia, eye findings, cervical adenitis (angle of jaw), in the presence of normal middle ears, a diagnosis of primary jugular bulb thrombosis was made. There was no evidence of any suppuration in the middle ear at any time. On March 22, the jugular vein was ligated, the general condition of the patient not permitting further surgical interference.

On March 24, the right lateral sinus and bulb were explored. The antrum and sinus plates were normal. The sinus was exposed in its mastoid course and for 2 inches behind the knee. On opening the sinus, an escape of pus was seen from bulb to torcular. There was no free bleeding. The blood cultures, three hours after operation, showed 20 colonies per c.c., and 48 hours after operation the blood culture was sterile and remained so. The temperature came down and remained normal. The papilledema gradually receded until on discharge there was a retrogressive neuritis. Patient was discharged Sept. 14, with healed mastoid and cervical scar.

Dorothy S., age 21 years; admitted March 8, 1922; discharged May 5, 1922. This patient was admitted to the Otological Service of Mt. Sinai Hospital, with a history of a chronic otitis media which had been discharging for many years. Following a sore throat, three weeks ago, the right ear became painful. The pain lasted for a week, and then subsided. One week prior to admission, the patient began to have fever, chills and sweats, which were associated with frontal headache and pain on the right side of her neck.

On examination, patient appeared acutely ill. The right eardrum was flat, there was a large perforation at the umbo, in which there was a granuloma. The glands at the angle of the jaw on this side

were tender. The fundi showed choked discs, the right eye to the extent of 2 diopters, and the left, 4 diopters. Blood cultures demonstrated that the patient had a bacteremia due to a nonhemolytic streptococcus. At operation the mastoid was found to be sclerotic. The sinus wall was discolored. Upon incising it a partially organized purulent thrombus was seen. On removing the thrombus, a large amount of thick, yellow pus welled up from the bulb. There was bleeding from the torcular end. Following the operation, the patient developed metastatic abscesses of the forearm and thigh, and a periarticular infection of the ankle. The abscesses were incised and drained, while the ankle subsided spontaneously. The temperature dropped to normal, the wounds healed, the papillitis subsided. The girl began to gain weight and was discharged.

SUMMARY OF CASES.

Aural History:		Cases
Bilateral middle ear suppuration:		1
Acute purulent otitis media:		7
Acute otitis media (no otorrhea):		1
Chronic purulent otitis media, central perforation, mild acute exacerbation:		1
Type: Mild. Recent acute otitis media or resolving—History of repeated attacks of acute otitis media:		7

General Symptoms:	Cases	Ocular Fundi	Cases
Chills	4	Optic neuritis early	
Septic temperature	9	to advancing	3
Headache	3	Papillo-edema	2
Vomiting	2		
Spleen enlarged	3		
Liver enlarged	3		
Cervical glands enlarged and tender	4		
Cutaneous (macula-papular and postular)	5		

Operative Findings:			
Acellular (sclerosed)		8	
Mastoid cells (not involved)		1	
Sinus plate:	intact	9	
Sinus wall:	diseased	9	
Sinus contents:	purulent obturating thrombus	7	
	fluid pus	1	
	thrombus not demonstrable	1	
	bulb absent bleeding	8	
	torcular: No bleeding	2	
	torcular: Yes bleeding	4	
	torcular: Doubtful	3	(Free bleeding from superior petrosal)

POST-OPERATIVE COURSE.

Metastases and Complications:		
Five cases:	Tempero-sphenoidal abscess	1
	Pulmonary: Bilateral embolic process	1
	Joints and soft parts	3
	Sympathetic meningitis	1
Fundi: post-operative:	Four—negative.	
	Five—increased changes and then recession to normal.	
Outcome:	Eight cases recovered. One ceased.	

BLOOD CULTURES.

Before and after operation: Eight of the nine cases had a positive blood culture; seven, hemolytic streptococcus; one, nonhemolytic

streptococcus. One case, negative before operation, was positive after operation. Two cases were negative after operation and remained so. Seven cases were positive for a time after operation. In one case (J. S.) blood cultures were peristently positive for five weeks. (Recovered.)

Differential blood cultures were done in two of these cases. The first of these (A. S.) had a bilateral otitis media; the problem of which side to operate upon, therefore, confronted us. These cases had a marked preponderance of bacterial growth in the blood cultured from the internal jugular vein on the sound side as compared with the blood taken from the vein on the diseased side. Similar findings were present in an additional case seen by Ottenberg in another hospital. One of our cases (not a primary bulb) at Mt. Sinai Hospital had a preponderance of bacterial growth in the blood obtained from the jugular vein on the *diseased* side.

As previously stated, these findings are indicative of the presence of a thrombus and suggestive of the direction or course of the blood invasion. Further studies must be made along these lines before formulating a definite statement as to their localization value in cases of bilateral middle ear or mastoid involvement. Case A. S. presented an acute bilateral middle ear suppuration in which an obturating thrombus was found on the left side. The differential blood culture had shown only a small number of colonies from the jugular vein on this side and a large number from the jugular vein on the healthy (right) side. Similarly, in the other cases (unilateral) showed a greater number of colonies grown from the blood obtained from the internal jugular vein on the normal side. In all of these an obturating thrombus was found at operation. In one case (not primary) a mural thrombus was found at operation. The differential blood culture in this case disclosed a far greater number of colonies cultured from the internal jugular vein on the *diseased* side than from the normal side.

Below is appended, in outline form, Ottenberg's differential blood culture findings in a number of cases. The first three were primary bulb cases (two reported in this paper). The fourth and fifth were cases of sinus thrombosis secondary to mastoiditis. The sixth was a case of bacterial endocarditis.

A. Cases of Occluding Sinus Thrombosis.

Name	Findings	Side	Occlusion	Organism	Colonies per cc. Rt. Int. Jug.	Lt. Int. Jug.	Periph.
(1) Stern	Sinus thromb.	L	+	Strepto-	278	9	
(2) E. M.	" "	R	+	coccus	15	67	
(3) Nuna	" "	L	+	Hemoly-	25	8	
(4) Grant	" "	L	+	ticus	122	7	6
"			Post-operative		225		22
"					356		78

				B. Case of Mural Thrombosis of Sinus.			
(5) Sontag	Sinus thromb.	L	O	Strepto-coccus non-hemolytic	16	142	
				C. Other Condition.			
(6) Sugarman	Bacterial endo-carditis	—	—	Strepto-coccus viridans	14	10	16

The fourth case (Grant—not primary bulb; unilateral otitis media purulenta and acute mastoiditis) is of unusual interest and will be reported at a later time. The differential blood culture findings were suggestive of the presence of an obturating thrombus. At operation an obturating thrombus was removed from the sigmoid sinus. Profuse bleeding and the poor general condition of the patient necessitated cessation of further operative interference. Two days later there was a recurrence of symptoms; additional differential blood culture reports confirmed the need for further intervention. At the second operation an obturating thrombus was found to have extended almost to the torcula.

CONCLUSIONS.

1. A mild middle ear infection in young children, associated with a septic temperature otherwise unaccounted for, suggests the possibility of a primary jugular bulb thrombosis.

2. For a favorable outcome, early diagnosis and prompt surgical interference, properly directed, is imperative.

3. Blood culture studies of Libman and more recently of Ottenberg materially aid in the diagnosis.

4. Occlusion of the main channels of systemic invasion, especially the torcular end of the diseased sinus, as suggested by Ottenberg's differential blood cultures studies, is of prime importance.

5. A jugular bulb operation should be performed only if there is a continuance of bacteremia and symptoms, general and local, after the main channels have been obliterated. Extensive jugular bulb operations are seldom indicated.

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12 East 86th Street.

THE UPRIGHT POSITION AND A VERTICAL RADIOGRAPHIC UNIT FOR ROENTGEN RAY EXAMINATION OF THE NASAL ACCESSORY SINUSES.

DR. SIDNEY ISRAEL, Houston, Tex.

To the oto-laryngologist chiefly is the X-ray examination of the nasal sinuses and other parts of the head most important. Skilled interpretation, together with the proper technique of production, are the two essentials if we would obtain the maximum aid from this important diagnostic means.

My study of microscopic and surgical pathology of paranasal sinus diseases[1], together with the comparative X-ray and operative findings in each case, has brought about the opinion that there is a distinct advantage, unsurpassed by any other method in my experience, afforded by the radiograph examination of sinuses in the vertical or upright position.

The usual methods employed at the present time in producing radiographs of the head, accessory sinuses and mastoids, are either with the patient placed horizontally, face down on a table, or sitting at a table with the head bent or flexed on a head board, in order to obtain the necessary angles of position. Both of these methods especially when dealing with elderly or stout persons with short necks. possess certain drawbacks and limitations well known to all of us, Children, too, are easily frightened and difficult to examine when placed face down on a table, beneath a mass of apparatus suspended above.

The upright apparatus described permits of a radiograph being made of any patient able to sit or stand. The upright position is the same essentially as one assumes in having a photograph made, with the back turned to the camera.

Rapid placing of the patient in position and ease and comfort during the exposure is obtained. An added advantage is that a minimum of floor space for installation is required.

Fig. 1 is a view of the entire apparatus. The two upright or vertical posts are 76 inches high and placed 19 inches apart. To these vertical posts are attached two horizontal crosspieces. These crosspieces are adjustable and can be locked in any desired location, and

1. "Chronic Disease of the Maxillary Sinus; Diagnosis and Surgical Treatment." Dr. Sidney Israel, Houston, Tex. Archives of Oto-Laryngology, June, 1928, Vol. 7, No. 6, p. 565.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication July 30, 1928.

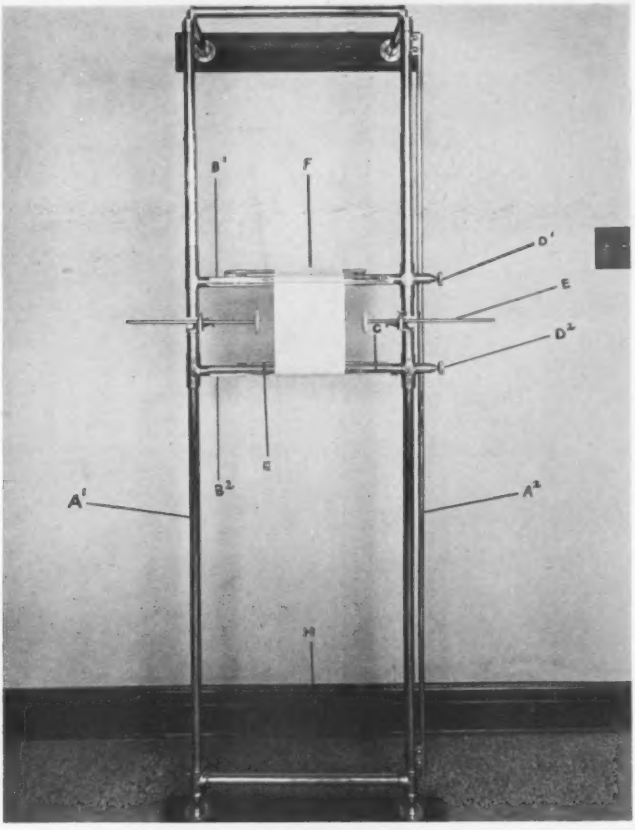

Fig. 1.

A1-A2. Vertical hollow uprights, containing counter-balanced weights.
B1-B2. Horizontal crosspieces, to which is attached:
C. Channel guide to hold cassette.
D1-D2. Lock nuts for B1 and B2, allowing horizontal crosspieces to be set at any height, from top to bottom of vertical uprights and adjustable so as to accommodate a 5x7 or 14x17 cassette. When so adjusted to move as one, to any desired level.
E. Removable head clamps.
F. Sanitary paper roll.
G. Rewind for soiled paper.
H. Foot rest.

The entire apparatus projects from the wall 9 inches and is counter-balanced, permitting of easy and rapid adjustment.

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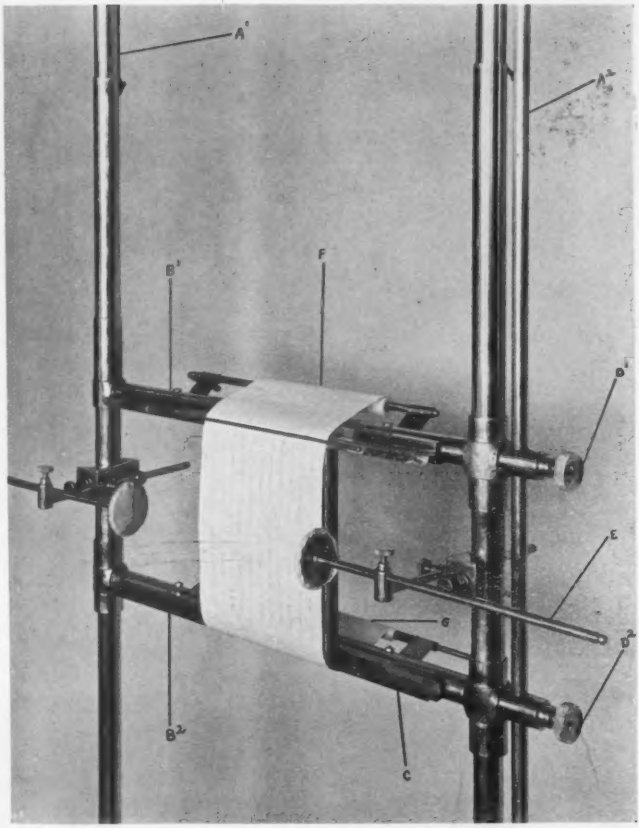

Fig. 2.

Close-up view of essential working parts of Fig. 1.

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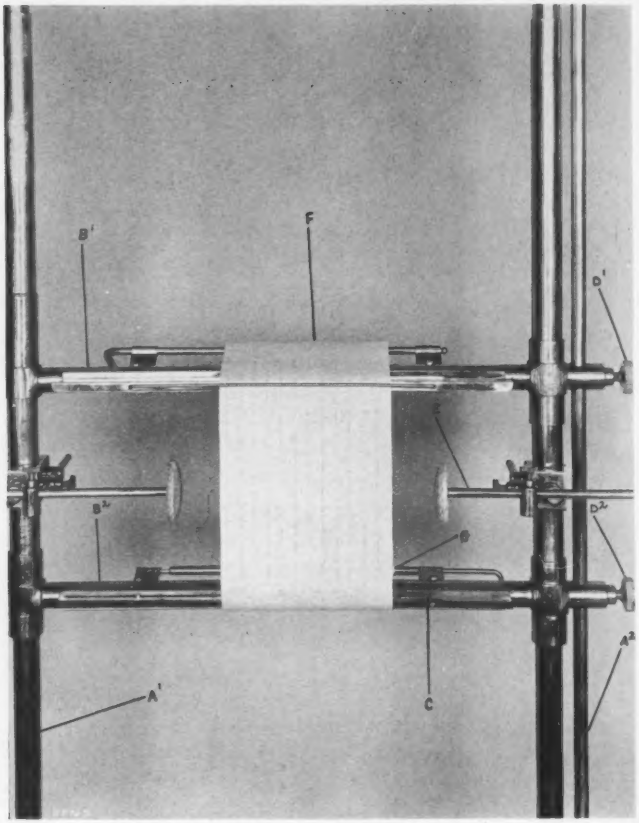

Fig. 3.

Close-up view of essential working parts of Fig. 1.

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can be moved toward or away from each other, so as to accommodate any cassette, ranging in size from 5x7 to 14x17 inches.

There is a channel guide in which the cassette slides and is held in position. The horizontal crosspieces are counterbalanced and can be moved as one, holding the cassette at the desired level opposite the site to be examined. The apparatus is so constructed that the horizontal crosspieces can travel from the floor to the top of the vertical parts, locking at any given elevation.

There is a paper roll that supplies a sanitary surface on the cassette for each patient's face, after each exposure. Head clamps that are

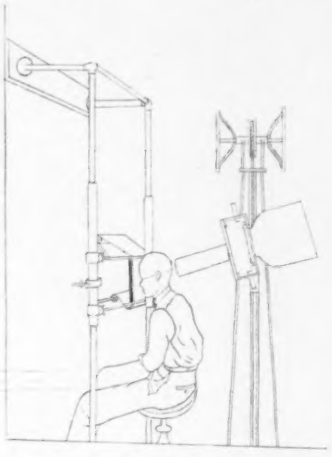

Fig. 4A.

Schema showing frontal, ethmoid, sphenoid positions, with central focus of the ray through nasofrontal articulation in upright position—nose and forehead on cassette.

easily and quickly removed or adjusted are utilized and likewise move as a unit with the horizontal crosspieces.

The entire apparatus projects from the wall 9 inches.

With a simple attachment to the horizontal crosspieces one is able to utilize the Bucky diaphragm. A cassette holder is also employed for rapid stereoscopic views.

The advantage to be obtained in radiograph examinations in the upright position of the chest and abdominal contents is quite well recognized at the present time. It is my opinion that the same view

should obtain in the production of radiographs of the nasal accessory sinuses, for the reason that in the *upright position,* and *with this apparatus* we have a means whereby it is *possible to demonstrate* the

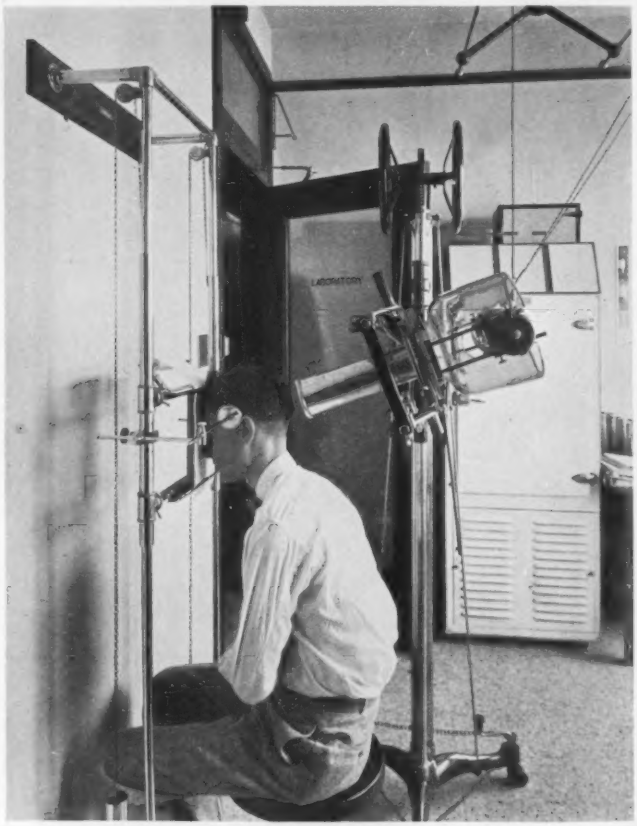

Fig. 4B.

Photograph of frontal, ethmoid, sphenoid—upright position.

level line when the sinus contains fluid or pus, an important aid in interpretation. This level line is clearly demonstrated in the upright position by means of lipiodol (Figs. 8A, 8B and 8C).

For the Visualization of the Frontal Sinuses, Ethmoids and Sphenoidal Sinuses in the Posterior-Anterior Position (Fig. 4C): The patient sits on a stool with the nose and forehead resting on the

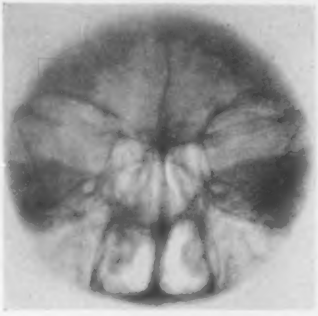

Fig. 4C.

Roentgen ray visualization of results obtained in upright position of Figs. 4A and 4B.

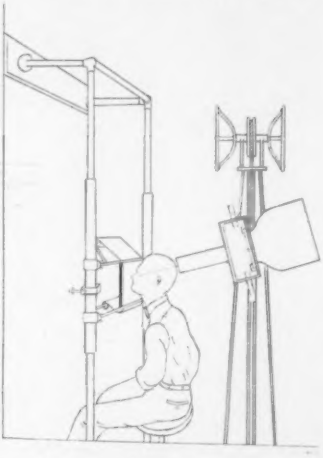

Fig. 5A.

Schema showing upright maxillary sinus position.
Central ray directed through a line ½-inch beneath outer angle of eyelids. Chin and nose resting on film cassette.

paper shield covering the film cassette, 5x7. The head clamps prevent mobilization. The central ray is directed through the root of the nose or the nasofrontal articulation.

Figs. 4A, 4B and 4C: This position and technique projects the frontal sinus and ethmoidal cells on the film, with the sphenoidal sinus slightly below the ethmoids, to avoid overlapping or masking one by the other.

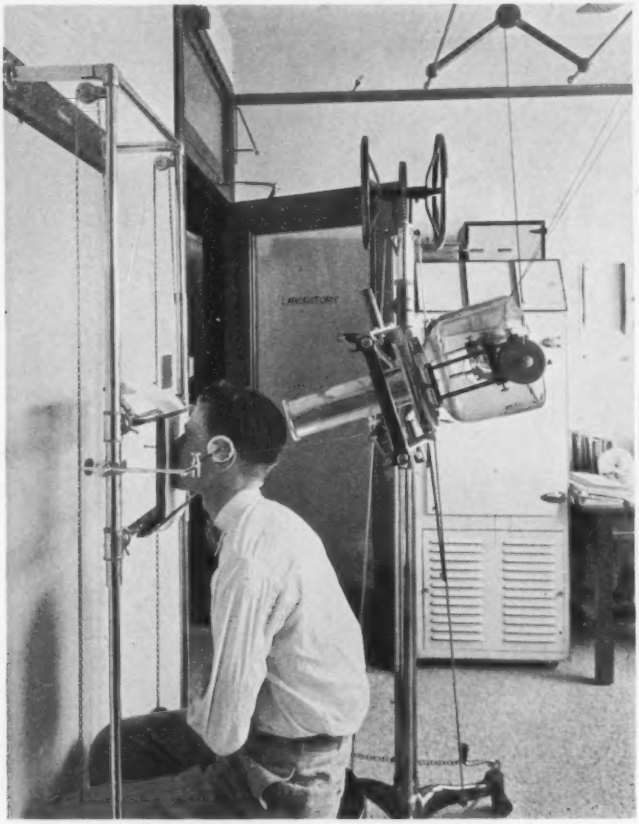

Fig. 5B.

Photograph of upright maxillary sinus position.

The Maxillary Sinus Position: Posterior-Anterior: Chin and nose on film cassette, head clamps for immobilization, central ray directed through a point ½-inch below external angle of eyelids.

Figs. 5A, 5B and 5C: The projection on the film shows an uninterrupted view of the maxillary sinuses, with the petrous portion of the temporal bone out of the way and below the level of the floor of the sinus.

With this apparatus and technique of focusing the central ray of the tube, the pyramid or petrous portion of the temporal bone is always projected below the level of the maxillary sinus, with a *minimum* of sinus *distortion.*

With the upright apparatus as herewith described the positions are quickly obtained. It is my opinion that fixed or arbitrary angles for X-ray visualization of the accessory sinuses are of limited value for the following reasons: *1.* The development of the head, facial contour and features vary practically with each individual and be-

Fig. 5C.

Roentgen ray visualization of results obtained in upright maxillary sinus position—Figs. 5A and 5B.

cause of these variations, when Roentgenographs are made at fixed or arbitrary angles, the end results when seen on the film also vary and are frequently unsatisfactory. *2.* The anatomical relationship of the nasofrontal articulation or root of the nose, with the location of the frontal sinus, ethmoidal cells and sphenoidal sinus is always the same, and serves as the surface landmark for the central focus of the ray by which is obtained satisfactory visualization of these sinuses on the film, with a minimum interference from overlapping. With the nose and forehead resting on the cassette, a satisfactory Roentgenograph is neither dependent on the size or shape of the head, nor differences in facial contour, provided the central ray is directed through the root of the nose.

A dependable landmark exists, namely, a point approximately ½-inch below the outer angle of the eyelids for the focus of the central ray to produce a satisfactory radiograph of the *maxillary*

Fig. 6A.

Showing upright position obtaining lateral view of head.

sinus, without interference, for the reason that the relationship of the maxillary sinus with the lower orbital border is always the same, regardless of facial contour and development.

Any method that permits of rapidity and ease in placing the patient in the desired position, together with the uninterrupted or unmasked projection of the sinuses on the film or plate, uninfluenced by differences in facial contour or development, and occupying a minimum of floor space, approaches the ideal.

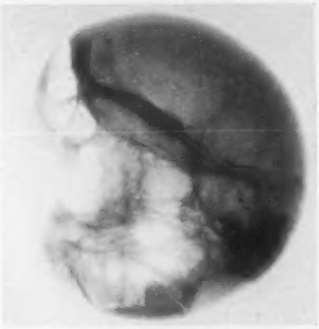

Fig. 6B.

Showing lateral view of frontal, ethmoids, sphenoidal and maxillary sinuses, in upright position.

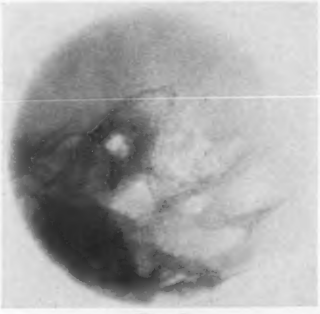

Fig. 7.

Roentgen ray examination of mastoid, with patient in upright, sitting position.

Because of the simplicity and ease in producing identical and accurate Roentgenographs in each or the same case, and *ability* to *demonstrate a level line* when present, the upright position and apparatus has been a most valuable aid in our daily routine.

2001 Niels Esperson Building.

Fig. 8A.

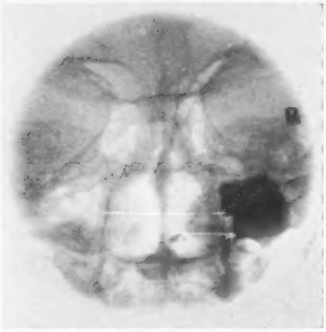

Fig. 8B.

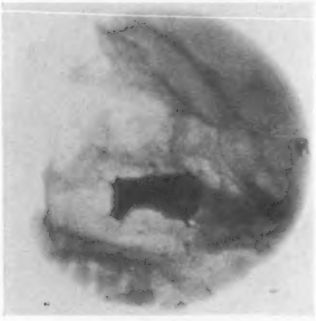

Fig. 8C.

Lipiodol injection of right maxillary sinus.
The level line or summit of the fluid is clearly demonstrated—also the irregular outline of the fluid content brought about by the alteration in the mucosal lining of the sinus.
Clinical diagnosis: Chronic maxillary sinusitis.

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CERTAIN ASPECTS OF TINNITUS, PARTICULARLY TREATMENT.*

DR. ISAAC H. JONES and DR. VERN O. KNUDSEN,
Los Angeles.

We all feel a general pessimism as to the prognosis in cases of tinnitus. If any otologist were to ask another otologist as to what success he has had in the treatment of tinnitus, he would not regard the answer as entirely facetious if it were, "I remember a case once that I think was cured". It is certainly true that none of us can look back on the cases of tinnitus that we have seen and treated, with a feeling that in any way approaches satisfaction. At times we have all felt, and no doubt the patient would particularly share this feeling, that, as Gray[1] stated regarding the theories of hearing, "One sometimes wonders if the mental energy spent upon the problem would not be better employed in some more fruitful research". However, if we had tinnitus ourselves, we would no doubt be grateful for any earnest attempt to understand and relieve it. No matter how hopeless certain cases may be, any sufferer from tinnitus at least wishes to try all possible means of relief before concluding that nothing can be done. Not infrequently we find that tinnitus may become less severe, and in rare instances may disappear altogether, apparently as the result of carefully selected and appropriate treatment.

The very multiplicity of the theories and treatment on the subject of tinnitus makes it appear that tinnitus still belongs, to a great extent, to the realm of speculation. Nevertheless, it is a problem that is ever present in the offices of the practicing otologist. Consequently this problem, although so largely speculative, requires not only the most careful consideration and study, but also systematic investigation and research. It may be that frequent considerations of this subject may ultimately suggest a method of approach that will be fruitful. At present the otologist is almost entirely limited to the mere observation of the existence of tinnitus, and various conjectures as to its origin and nature. Only a meager amount of experimental work has been done. Such investigative work probably should commence with the production and study of tinnitus under

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experimental control; also, measurements of the precise pitch and loudness of a tonal tinnitus; also, determination of the more complicated character of tinnitus which is not tonal. If a tinnitus be described as a noise like the "breaking of waves at the seashore", it may be possible to correlate this with a disturbance of the blood circulation within or adjacent to the cochlea. Or, if a tinnitus be described as "a rushing of air", there actually may be pneumatic disturbances in the conducting mechanism. Apparently it is only through such research work that the hope can be realized of having tinnitus emerge from the realm of speculation.

"Noises in the head" are not only the *bête noire* of the practicing otologist, but, much more important, they constitute one of the most distressing and persistent symptoms that can disturb a patient. The patient may be merely annoyed by a slight noise, perhaps transient in character, or he may be distracted by continuous and intense noises from which he cannot escape. As a human problem, therefore, tinnitus not only plays a part in minor nervous disturbances, but may result in insanity or suicide. Therefore, not only every otologist, but every intelligent patient has wondered and speculated as to what tinnitus actually is, what causes it, what disturbance is going on and where, what structure or structures are involved, not to mention what can be done to alleviate it. One speculates, for example, as to what is the actual lesion that occurs during the development of otosclerosis, whereby tinnitus is produced; what is the mechanism of the production of tinnitus in cases of impaired tubal ventilation, or chronic adhesive processes in the middle ear; and why it is that tinnitus results in cases of chronic degenerative processes in the internal ear. In any given case, if one can determine the mechanism of the tinnitus, a clue is at once given as to the approach toward the treatment. Even to locate the lesion responsible for the tinnitus is a step in the right direction.

MECHANISM OF TINNITUS.

Although the subject of tinnitus is most baffling, yet it would appear that there are certain things that we can understand. Apparently we can be sure of one thing: tinnitus is a disturbance of the auditory mechanism. Unless this disturbance affects the cerebral auditory centers, there can be no tinnitus. Although the tinnitus exists only in the auditory centers, the source may be not only within the centers, but also along the auditory pathways or in the auditory end organs. Although it is unusual for intracranial organic lesions to produce tinnitus, yet tinnitus has frequently been observed in cases of general mental disturbance which include auditory hallucina-

tions. It appears, however, that cerebral lesions or mental diseases are not necessarily the only factors that can produce permanent changes in the auditory centers; it has frequently been observed that the severing of the eighth nerve, or the destruction of the cochlea, has failed to relieve the tinnitus. This suggests that the repeated or continuous insults to the auditory centers have brought about permanent changes in the auditory centers, in the form of what one might term "memory pictures".

There is very little evidence of tinnitus produced by lesions along the auditory intracranial pathways. Lesions of the eighth nerve, however, may exhibit tinnitus. In the great majority of all cases of tinnitus, the exciting cause apparently lies within the end-organ itself, including the cochlea and the conducting mechanism of the middle and external ear. It seems reasonable to assume that the nervous mechanism of the end-organ can be disturbed in only two ways: first, by actual irritation or stimulation of the cochlear structures themselves; or, second, by the introduction into the cochlea of *vibrations* set up in the conducting mechanism of the middle ear.

First, considering the mechanism of the production of tinnitus, due to a lesion in the cochlea; a lesion involving the cochlea manifests itself in an impairment of hearing, or in tinnitus, or both. Frequently, one observes the hearing impairment without the tinnitus, and also, although less frequently, there may be a tinnitus with no impairment of the hearing. In fact, one might say that a lesion of the cochlea, or any disturbance of the cochlea, may result in two disturbances of function: impairment, or loss of the function of hearing, and perversion, as it were, of this function, which we call tinnitus. There is, of course, the other phenomenon of diplacusis, which might also be classified as a perversion of the hearing function. However, the two phenomena which are usually found in a cochlear lesion, are impairment of hearing and tinnitus. Diplacusis is relatively rare. In a case of actual lesion of the cochlea, a hearing impairment is always present, tinnitus frequently present, and diplacusis only rarely present.

It is a common observation that tinnitus is frequently associated with a hearing impairment. Not infrequently it happens that a patient first notices tinnitus and then, upon examination, it is discovered that he has an appreciable defect of hearing. In cases of tinnitus one is usually able to demonstrate the presence of a lesion, in either the conductive or perceptive mechanism. Occasionally, individuals with normal hearing are disturbed with a tinnitus; however, in such cases the tinnitus is not persistent.

Of course a tinnitus of any type will produce a masking effect, and this of itself would interfere with normal hearing. Whether a tone is produced from the outside or from a disturbance within the ear itself, as in cases of tinnitus, it seems only reasonable that the same type of masking effect would result. If the tinnitus is tonal in character, the masking effect would be greater for tones in close proximity to the tone of the tinnitus. Precise measurements of the hearing acuity of an individual exhibiting this type of tinnitus shows an actual loss of acuity for tones in the vicinity of the tonal tinnitus. On the other hand, if the tinnitus is lacking in tonal character, and is of the nature of a noise, involving an extensive range of tonal components, the masking effect is exhibited as a loss of hearing acuity over a wide frequency range. Minton[2] has shown that cases exhibiting tonal tinnitus actually show a loss of hearing acuity for tones which are in the proximity of the tonal tinnitus. Thus, if an individual has a tonal tinnitus corresponding to a frequency of 4,000 d.v., precise measurements demonstrate a loss of acuity or a tonal depression for tones in this neighborhood. This is comparable with objective masking. A subjective tone would be expected to have the same masking effect as an objective tone. Whether objective or subjective, a masking tone has its greatest interfering effect upon tones in the immediate proximity of a tone of its own frequency, but it also has the characteristic of interfering more with tones of a high frequency than those of a lower frequency. For this reason low pitched tinnitus affects a greater tonal range than a tinnitus of a high pitch. In any case of tonal tinnitus, the hearing acuity would be interfered with because of the lesion itself, primarily, but, in addition, there would be a masking effect of the tonal tinnitus which would further impair the hearing for tones in the proximity of the pitch of the tinnitus. For example, a lesion may be found in which there is a marked loss of acuity for tones in the vicinity of 4,000 d.v. It may be found that the loss of acuity in this range amounts to 50 per cent. The lesion itself might be responsible for perhaps 40 per cent of this hearing loss, the remaining 10 per cent being attributable to the masking effect of the tonal tinnitus.

The difficulty is to visualize what the nature of the disturbance may be that causes tinnitus. It would seem that it must be an irritation; but one can merely speculate as to whether this irritation be caused by congestion, inflammation, obstruction, increased pressure, adhesion or other mechanical interference. Nor do we know which cells or structures produce tinnitus.

TINNITUS EXCITED BY COCHLEAR LESIONS.

It would seem probable that there are many forms of disturbance of the cochlea that can result in tinnitus. Obviously, however, it must be some form of disturbance that causes an excitation of the hair-cells, or terminal nerve fibres associated with Corti's organ. The lesion that produces tinnitus must possess certain characteristics, and the lesion that produces deafness must possess other and different characteristics. This is suggested in that we observe some cases with tinnitus and others with hearing impairment. It may be that the same lesion will possess both sets of characteristics. The characteristic required for the production of tinnitus is that of excitation of the nerve fibres; the characteristic which produces an impairment of hearing would be the absence of a normal excitation of the nerve fibres. In this sense, a hearing impairment would be due to an insufficient nerve stimulation, and tinnitus would be due to an abnormal type of nerve stimulation—a perversion. On the basis of the Neuron theory, the loudness of a particular sound is determined by the number of neurons which are activated. Any lesion that would interfere with the excitation of the normal number of neurons would result in impairment of hearing. The impairment of hearing may result, for example, from imperfect articulation between the active membrane and the nerve endings. In such a lesion the membrane would have to be set into a relatively greater amplitude of vibration to affect a given number of nerve endings than would be the case with normal hearing. In the case of tinnitus, it is conceivable, for example, that the articulation between the active membrane and the nerve endings is more or less permanent, so that there is a continuous excitation of the nerve endings. In the case of tonal tinnitus this articulation would be limited to a small portion of the cochlear membrane. In the case of "generalized" tinnitus—that is, the tinnitus which is characterized by a noise—this abnormal articulation embraces a large portion of the active membrane. In brief, in cases of cochlear lesion, an impairment of hearing may result from inadequate articulation between the active membrane and the contiguous auditory nerve endings; whereas, tinnitus might result from exactly the opposite condition—a continuous coaptation of the active vibrating membrane and the auditory nerve endings, resulting in continuous stimulation and auditory impulses.

If the lesion is in the cochlea, the tinnitus may consist of a low pitched tone, a high pitched tone, or a noise, depending upon the location and extent of the lesion. Thus, on the basis of the resonance theory, if the lesion be, for example, in the first turn of the cochlea,

the tinnitus will be one of high pitch. If the lesion, or disturbance, be in the apex of the cochlea, the tinnitus will be one of low pitch. If the lesion extends over the greater portion of the cochlea, the tinnitus will consist of a general noise.

TINNITUS EXCITED BY CONDUCTIVE LESIONS.

It is admittedly simple to account for tinnitus when there is an actual irritation, or lesion, in a portion of the cochlea. For that matter, it is merely necessary to excite or stimulate any sensory end organ, in order to produce a characteristic sensation, whether tactual, olfactory or auditory. However, it is not such a simple matter to account for tinnitus in cases in which the cochlea is normal, and the only demonstrable lesion is in the middle or external ear. Although we know that tinnitus may be caused by a plug of wax, or a middle ear lesion of almost any type, if such a lesion does not affect the cochlea, there can be no tinnitus. Obviously, a lesion within the middle ear could not of itself produce tinnitus—that is to say, adhesions or other form of lesion in the middle ear would not, of themselves, produce an irritation of the auditory endings, and therefore, it would seem that whenever such a lesion in the middle ear produces tinnitus, it would be due to the introduction of vibrations into the cochlea. Naturally adhesions or obstructions would not, *per se,* set up any vibrations, and without vibrations it would be difficult to account for the production of tinnitus from such a middle ear lesion. It is easy to understand the conduction through the middle ear of vibrations from the outside world. It is not so easy to understand the creation within the middle ear of vibrations. It would seem that the only structures within the middle ear that could cause alternating contractions are the muscles of the middle ear.

A study of the physiologic actions of any muscle includes the observation that the contraction of a muscle does not consist of a steady pull, but possesses a periodic or vibratory character. Many laboratory courses in physiology include the demonstration of the contraction of a preparation of the muscle of an experimental animal, usually the frog. The muscle is stretched and attached to a recording stylus, so as to give both an amplification and a record of the motion. For example, when the muscle preparation is irritated, or stimulated, in any way, the recording stylus shows that the contraction is made up of a series of periodic pulls.

Based upon this known fact as to the physiologic action of muscle, Lathrop[3] has advanced an ingenious theory. He considers that a disturbed ventilation, or chronic adhesive processes, or a displacement of the drum membrane, as, for example, by a plug of wax in the

external canal, or any other factor that might cause the displacement of the ossicular chain, destroys the normal balance existing between the ossicular chain and the two middle ear muscles. In this way, either the tensor tympani muscle, or the stapedius muscle, or both, are in a state of tension. According to this theory, the muscles attempt to restore a condition of balance, such as normally existed before the adhesions, or other processes, disturbed the ossicular chain. Lathrop suggests that the tensed contractions of these muscles attempt to restore the ossicles to their normal position. He further suggests that the contractions of these muscles reasonably would be supposed to be of a vibratory nature. These vibrations would be communicated to the cochlea, resulting in tinnitus. This theory of Lathrop, therefore, gives a plausible explanation as to the causation of vibrations generated within the middle ear itself, and not coming from the outside world. The periodic contraction of the middle ear muscles themselves could furnish such vibrations.

In considering this theory, it is necessary to inquire whether such a state of unbalance of the ossicular chain could reasonably be produced by the well known external and middle ear lesions which do produce tinnitus. It would be reasonable to assume that these well known lesions could produce such an unbalance. For example, a plug of wax in the external ear, pressing against the drum membrane, could cause a displacement of the drum membrane and consequently of the ossicular chain. It seems equally reasonable that disturbances of ventilation within the middle ear due, for example, to tubal obstruction could produce such a displacement by means of the difference between the atmospheric pressure in the external air, and the pressure within the middle ear. Likewise, chronic adhesions, whether due to chronic catarrhal processes, or to previous suppuration, with resultant contractures, or actual arthritis in the joints of the ossicular chain, or even the overgrowth of tissue around the stapes head in otosclerosis—all of these can similarly displace the ossicular chain, in some portion, from its normal position. These displacements from the normal position are opposed by the muscles, tensor tympani and stapedius. We know that tinnitus does occur in cases with such lesions, and yet, as stated above, we also know that in order to have tinnitus, there must be impulses initiated either within the cochlea, or conveyed to the cochlea in the form of vibrations from without.

Further, it would seem possible that the middle ear muscles reasonably could be expected to vibrate with a sufficiently high frequency to produce vibratory stimuli within the tonal range. The rate of vibration of the contracting muscle would depend in large measure

upon its size—the smaller the muscle, the more rapid the rate of vibration. In like manner, the greater the tension of the muscle, the greater the rate of vibration. Since these muscles are so very small, and are in a state of at least average muscular tension, it seems reasonable to suppose that their rate of vibration would certainly be high enough to be in the audible range. It is therefore plausible that the vibratory contraction of these muscles could result in tinnitus.

It is more difficult to conceive how these vibrations can be maintained so consistently and continuously to account for persistent tinnitus. Ordinarily, vibrations produced in this way are subject to considerable damping—that is, the amplitude of the vibration becomes smaller and smaller, until ultimately it disappears. Usually, the complete cessation of vibration in such muscular tissues would be expected to occur in a few seconds. Furthermore, in the case of the contracting muscles in the middle ear, one would expect that ultimately there would be established some form of balance or compromise between the opposing forces of, say, the adhesions on the one hand, and the muscle contractions on the other, with the result that the muscle contractions would cease, and consequently the tinnitus would cease. This, however, is not borne out by the facts. Tinnitus of this type lasts for months or years, and at times indefinitely. This explanation of tinnitus, due to middle ear lesion, would seem to satisfy the physical requirements, with the exception of accounting for the indefinite persistence of the muscular contraction. However, it seems necessary to advance some such hypothesis as this of Lathrop to account for a stimulation of the cochlea with a production of tinnitus, when the lesion itself is in the external or middle ear.

TYPES OF TINNITUS.

It is really of no importance at all, clinically, whether we classify tinnitus in one way or another. Therefore, such a classification will be made only because it affords the opportunity to discuss certain really important characteristics of the different types.

One is confronted with difficulties in attempting to classify the different types of tinnitus; it does not lend itself to any hard and fast classification. Various classifications could be utilized, depending entirely upon the angle or point of view with which one regards the subject. Tinnitus may be classified as to its origin and nature; the location of the essential disturbance or lesion, whether in the conductive or perceptive apparatus; whether entirely subjective or objective—in which latter case it is not only heard by the patient, but by another observer; with reference to whether the tinnitus is tonal or

consists of a general noise; whether the tinnitus is or is not associated with a hearing impairment; whether the tinnitus is continuous or intermittent; finally, whether the tinnitus is due to a peripheral cause or is central in nature, as in psychic tinnitus consisting of hallucination.

As to the origin of tinnitus, an inquiry into the history of the patient, and an examination may reveal that the tinnitus was caused by acute or chronic conditions within the middle ear; otosclerosis; toxemias, such as those of mumps, scarlatina, influenza or syphilis; following the use of certain drugs, such as quinin and the salicylates; thyroid deficiency or hyperthyroidism; neuroma of the eighth nerve; associated with a low blood pressure or high blood pressure; or associated with pregnancy, either during or after the pregnancy.

The most usual clinical classification of tinnitus is on the basis of the location of the lesion, whether in the conductive or perceptive apparatus. In general, lesions of the conductive mechanism may exhibit a variability in the tinnitus, whereas lesions of the perceptive mechanism, as a rule, exhibit tinnitus of a continuous and more or less constant nature. Usually a tinnitus due to a lesion of the conductive mechanism may be influenced by changes in the weather, conditions within the nose and throat and other similar causes. Lesions of the internal ear are not apt to be so affected. On the other hand, the lesions of the perceptive apparatus are affected by such conditions as general nervous fatigue.

Another classification—that of subjective and objective—brings in another feature of tinnitus. In objective tinnitus the source is outside of the ear itself. Such noises are merely conducted to the ear through contiguous structures. These are really external noises or physicallly produced sounds, which may occasionally be heard by someone else besides the patient himself. Of course this is not a true tinnitus and might be termed a pseudotinnitus. One of our patients exhibits a spasmodic contraction of the palatal muscles, unilateral, which causes a clicking sound, presumably because the Eustachian tube is forcibly opened with each contraction. This sound can also be heard by the observer by putting his ear near the ear of the patient.

All sounds may be classified as either tone or noise. It is an observed fact that tinnitus, in a similar way, may be classified either as tone or noise. The tinnitus which sounds to the individual as a more or less pure tone may be spoken of as tonal tinnitus. As a rule, however, the tinnitus is not tonal and is more often described in

general terms of noise. One hears descriptions of every sort, with resemblance to the actual tones or noises with which we are familiar in our normal auditory experience. Some of the most common expressions of patients include the "roaring of the ocean", "falling of water", "chirping of crickets", "escaping of steam", or "the rushing of air". The descriptions of these noises are so varied that one might almost say that each patient has his own particular phrase or idea or analogy with which he describes his tinnitus. It is not yet possible to correlate the nature of these sounds with any particular lesion responsible for them. There is no tinnitus characteristic of a conductive lesion, nor is there any particular type of tinnitus characteristic of a cochlear lesion. In general, one finds that a conductive lesion may occasionally be associated with a tinnitus of fairly low pitch, although many conductive lesions do exhibit lesions of high pitch. In cochlear lesions the tinnitus is almost always of high pitch; not very infrequently the tinnitus is tonal in character.

Tinnitus might again be classified as tinnitus associated with a hearing defect, or without a hearing defect. However, in a case of tinnitus that is really disturbing and continues for weeks and months, it is rare to find that a hearing defect cannot be demonstrated in some portion of the tonal range. Continuous severe tinnitus, therefore, is seldom seen in individuals with perfect hearing. In sharp contrast, tinnitus is frequently observed in cases in which apparently there has been a complete destruction of the hearing function. *A patient:* Basal skull fracture; examination showed right ear uninvolved, but left ear entirely without function, either cochlear or vestibular, yet his one great distress was the tinnitus in this (nonfunctioning) left ear. *A patient:* C. H. Frazier had severed the eighth nerve; tinnitus ceased; then recurred after one year; caloric test gave no response, showing that the eighth nerve had apparently been completely severed, yet the tinnitus returned. *A patient:* Almost suicidal; Frank Kistner did an extirpation of both cochleas; tinnitus disappeared for approximately six weeks, but then it began to recur in the left ear, gradually grew more intense; at the end of two months the tinnitus began in the right ear; when last seen the patient was suffering a great deal from tinnitus in both ears, although not so bad as before the operation.

Perhaps the best explanation for such cases is that suggested by Richard Lake (The Lancet, May 30, 1925): "After tinnitus has been peripheral for a certain length of time, the outside duration of which I would myself place arbitrarily at three years, the subjective sound becomes central . . . that is to say, a prolonged irritation of the

central ganglion cells has caused the ganglion cells themselves to become diseased or disorganized, and the tinnitus becomes central."

THE PREVALENCE OF TINNITUS.

Fortunately tinnitus is not a necessary accompaniment of hearing impairment. Naturally, all lesions affecting the conductive or perceptive mechanism of the ear, necessarily result in a hearing impairment; they do not necessarily result in the symptom of tinnitus. However, in an analysis of 340 cases which exhibited a hearing impairment, varying from a slight impairment to total deafness, tinnitus was complained of, or noted, in 201 cases, or 59 per cent. Of these 201 cases of tinnitus, 70 were associated with conductive lesion; 54 with perceptive lesion; and 77 with mixed conductive and perceptive lesions.

Of the 340 cases of hearing impairment, 111 cases classified as conductive; 95 as perceptive; and 134 as mixed conductive and perceptive.

In the 111 cases of conductive impairment, tinnitus was observed in 70 cases, or 63 per cent. In the 95 cases of perceptive impairment, tinnitus was observed in 54 cases, or 57 per cent. In the 134 cases of mixed conductive and perceptive impairment, tinnitus was observed in 77 cases, or 58 per cent.

The cases in this series might be regarded as typical and representative, as they were all seen in routine office practice. The significant findings in this survey are: *1*. The surprisingly high prevalence of tinnitus—59 per cent. *2*. The particularly high prevalance of tinnitus associated with conductive lesions—63 per cent. *3*. The general uniformity of the prevalence of tinnitus in different types of deafness—of the total, 59 per cent; of the conductives, 63 per cent; of the perceptives, 57 per cent; of the mixed conductives and perceptives, 58 per cent.

TREATMENT.

Classification: Local treatment of the patient; general treatment of the patient; use of mechanical devices. Local treatment is limited to conditions of the external and middle ear; removal of obstructions in the external ear; varied measures to improve the condition of the middle ear, such as the standard surgical procedures in the nose and throat, and local treatment to the Eustachian tube and middle ear. Such measures are not altogether unsuccessful. We have in mind a woman who had persistent tinnitus in both ears for two years; tonstillectomy; completely relief, now lasting three years. This woman also had a hearing defect, which was not improved, although we had

reason to think that it was arrested, as up to that time she had been getting more and more hard of hearing. Precise measurement shows the same picture today as that of three years ago. Another woman had tinnitus, which she stated was "unbearable"; tonsillectomy was followed by relief of tinnitus in a very short time; also improvement of hearing in the right ear from an average of 30 per cent to an average of 50 per cent. Our "successes", however, have been pitifully few. Most otologists have found that the best prospect of local treatment is in cases of tubal congestion. Lake[4] has noted relief of tinnitus by cautious intertubal injections of glycerin-acid-carbol. Ruskin[5] reports cessation of tinnitus in two cases, following cocainization of the sphenopalatine ganglion.

Several of the best otologists have severed the eighth nerve or destroyed the cochlea, in cases in which the tinnitus has been distracting. Nearly all now have concluded that such operations are not without danger, and that the results are almost always unsatisfactory. Lake[6] expresses it, "As I have already stated, my contention is that in the future fewer operations for the relief of tinnitus should be performed. I have myself destroyed the cochlea in five instances, with one questionably good result. Other forms of operation have been advocated, and there have been a large proportion of fatalities. There is, practically speaking, not one proven cure."

We have all utilized many methods of local treatment—it is possible that many of us have been less alert than we should be in the use of constitutional treatment, directed toward the general condition of the patient. Focal infection, or constitutional disorder, may produce disturbances in the middle ear, but much more frequently affect the internal ear. General treatment, therefore, is usually directed toward the cochlea. Experience has caused many of us to consider that cochlear defects are frequently the results of a focus of infection, the tonsils, teeth, nasal sinuses, or in even more remote locations, such as the gall bladder or prostate. Any marked cochlear defect, with or without tinnitus, should make us search diligently for a focal infection, particularly if we cannot find any other cause of a previous toxemia, such as mumps, scarlatina, influenza or meningitis. Toxemias from such diseases have already produced the damage in the cochlea, or eighth nerve, and naturally, there is no treatment. It is a different matter if a cochlear defect, with or without tinnitus, leads us to discover syphilis. Treatment is instituted, although unfortunately such treatment usually does not relieve the cochlear defect or the tinnitus. We find cases in which drugs, particularly quinin, have produced a cochlear defect and tinnitus, and in these

cases it is also usually true that the damage has been done and no definite treatment is possible. Alcohol and tobacco at times appear to increase a tinnitus, in which case the patient is naturally advised to avoid them. Certain drugs have been empirically recommended, particularly by English otologists, which have relieved tinnitus—notably dilute hydrobromic acid in doses of one fluid dram three times a day, in water; this may be combined with small doses of potassium iodid.

In cases of hyperthyroidism, suitable treatment (such as Lugol's solution and rest), at times relieves the tinnitus which occasionally accompanies this condition. Certain otologists report good results from the use of thyroid extract, in cases of hypothyroidism exhibiting tinnitus as a symptom. Similarly, tinnitus observed during the menopause may be relieved by ovarian extract.

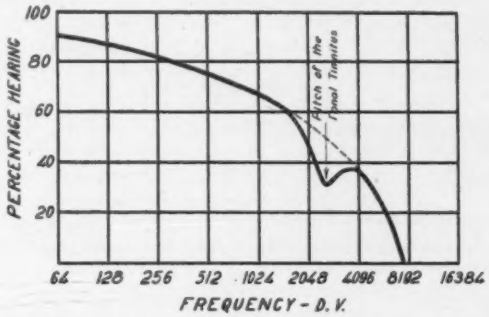

Fig. 1. Auditory chart showing the masking effect of a tonal tinnitus in a case of perceptive impairment. (The solid-line curve shows the measured percentage hearing. The dotted line indicates what the percentage hearing would probably be in the absence of the tonal tinnitus.)

Many clinicians have regarded blood pressure as an important factor in tinnitus. It is certainly advisable to investigate the blood pressure in all cases of tinnitus. If the systolic pressure is low, such drugs as digitalis or strychnin may give relief, by elevating the systolic pressure. In cases of high blood pressure, the English otologists particularly have recommended trinitrin. Naturally, such conditions are best cared for by the internist. In cases in which the blood pressure appears to be a factor, the tinnitus is usually in both ears. Reik[7] reports a case of complete relief from severe tinnitus, unilateral, by ligation of the common carotid artery; in another case, a small aluminum band was placed so as to constrict the common carotid artery, with resulting relief of the tinnitus.

Mechanical devices. We have constructed two types of instrument: One, for what might be termed the "bombardment" of the cochlea, and the other type for the purpose of masking the tinnitus.

The "bombardment" was suggested by the animal experiments of Siebenmann ,Wittmaack and Yoshii. The object is to desensitize the cochlea in certain tonal regions—the regions corresponding to the tinnitus. The pitch and loudness of the tinnitus are first determined by precise measurements. An objective sound is then produced, of the same pitch as the tinnitus but much greater loudness. In this way, by bombarding the cochlea with excessively loud tones, we have at times noted lessening of the tinnitus. Unfortunately, this fatigue phenomenon, which it undoubtedly is, does not last.

The other type of instrument is for the temporary masking of tones or noises. Two different instruments of this type may be used. The first instrument is a monotone generator. Such a generator can be

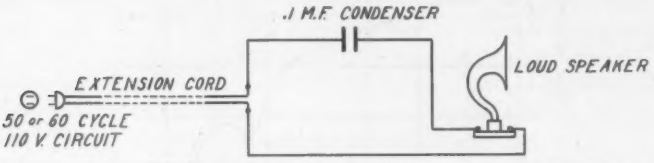

Fig. 2. Circuit diagram of a Harmonic Generator used for masking tinnitus. The 0.1 m.f. condeser emphasizes the higher harmonics. By introducing an overloaded transformer in the line, the higher harmonics can be further emphasized.

readily constructed of an appropriate vacuum tube oscillator and a loud speaker. The oscillator is adjusted to produce a frequency slightly below the frequency of a tonal tinnitus.

The other instrument might be termed a harmonic generator. The instrument is adapted for masking tinnitus which is characterized either by a tone or a noise. In order to do this, the instrument must produce a complex "spectrum" of tones, with a preponderance of low pitched tones. These requirements were obtained by a very simple device. An ordinary 50 or 60 cycle alternating current is conducted through a special loud speaker and a telephone condensor connected in series. The loud speaker is a Magnavox type with an adjustable diaphragm. The adjustable diaphragm provides a convenient means for controlling the loudness of the sound. The telephone condensor of one-tenth microfarad capacity, offers a high impedance to the fundamental 50 or 60 cycles, but offers progressively less impedance to the harmonic overtones which accompany it. In this way, the

higher harmonics are relatively emphasized. As a result, the current actuating the loud speaker is rich in the higher harmonics and consequently the loud speaker produces an entire harmonic series or "spectrum" of tones. The higher harmonics are further emphasized by overloading the loud speaker. This produces additional distortion, or an increased prominence of the higher harmonics. In this way, there is obtained a broad series of frequencies, extending throughout the important portion of the speech and music range. This provides a type of sound which is capable of masking almost any tone or noise which may characterize a tinnitus. Incidentally, this "blanket noise" gives a patient with conductive impairment a relative hearing advantage over the person with normal hearing.[8]

Although, of course, such an instrument does not "cure" the tinnitus, yet we may find after suitable experience that relief may be provided for those, for example who are unable to sleep because of their tinnitus. A patient is not distressed by a sound which is produced outside of himself.

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1920 Wilshire Boulevard.

TREATMENT OF X-RAY AND RADIUM BURNS.

DR. J. EASTMAN SHEEHAN, New York City.

While there is a very strong temptation to generalize about these X-ray and radium burns, probably it will best meet the needs of the present occasion to cite the particulars as to a certain number of cases, afterwards noting any lesson that it may be practicable to learn from such conditions as may be more or less common to all of them.

1. The first is that of a young woman who had a very extensive angioma of the right side of her face. She had been treated for years in the belief that rays and radium emanations would control the tumor and perhaps reduce it to an inactive state. Trismus had developed when she came to me. The explorations made with a view to restoration of function revealed, first, that the tumor was apparently unaffected by the long series of treatments; and, second, that all the tissues, of every sort, surrounding the tumor had become completely devitalized. It was not merely the mucous lining of the mouth that had become contractile and produced the trismus; the whole cheek was in that state. This meant that extirpation of the tumor must be accompanied by excision of all the tissues surrounding it, and as the tumor had invaded the tissues almost to the orbit, and also the mouth and tongue, there was very little left of that side of the face when the dissection was finished. Had the extirpation of the tumor been made in the first instance there would have been healthy remainders, upon which to base reparative procedures; these had now been destroyed and rejected.

To furnish this patient with a substitute for the whole of one side of her face involved the employment of many expedients and the exercise of much patience by herself in presence of numerous disasters. The first flap used for a cover after the trismus had been released sloughed and left us no further ahead. However, the gap was ultimately covered, two leaves of skin giving a certain consistency, one leaf acting as lining and the other as cover. On this foundation several minor adjustments were made from time to time. Perhaps the most interesting was the insertion between the inner and outer leaves of bands of fascia, the upper ends of which were attached over the temporal muscle. The prime intention was to over-

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come the tendency of the simulated cheek to sag, but the response of the fascia to the motions of the temporal muscle, when eating, initiates motion which is suggestive of function.

2. A second case was that of a girl who had developed, when 6 months old, an ulceration that spread from her lip up over the cheek and nose to the forehead, not characteristic of any disease, but constituting a disfigurement that, as she grew older, isolated her from social contacts. She had been treated by a number of doctors, all of whom had believed that something might be done by treatments with rays and radium. She was referred to me when trismus developed, and here again examination disclosed that the dysfunction was due to perversion of the tissues resulting from the ray treatments. All this contractile tissue had to be cleared away, both inside the mouth and over considerable areas on the surface. A buccal inlay of epidermic graft restored the mobility of the mouth, which had been impaired, as to one part, by the adherence of the lower lip to the gingival surface. A double tubed pedicle graft effectively restored the function of the lip and chin. Several other reparative procedures were addressed to the disfigurements about the nose, forehead and canthus.

3. In a third case, the repair was by what in practice is the smallest of the skin grafts, that from the upper eyelid. It is mentioned here as a reminder that there is no single method of universal application. Every case is a separate problem demanding its own solution; there is no mass production in this kind of surgery.

The patient had a small hairy mole, near the angle of the mouth. There had been repeated X-ray treatments, leaving a mottled, discolored, highly pigmented area, with considerable sheen, which last was exaggerated under a reflected light.

After excision of the burned tissues, the defect was covered by a small graft from the skin of the upper eyelid. This implant is now well incorporated with the surrounding skin. The usual experience with these eyelid grafts is that they turn red at first, the color disappearing from the periphery inward. In this particular case the red deepened into brown, almost black, before the return to normal color began. Whether this was due to one of those peculiar pigmentations to which the lids are subject, or was a sign of delayed restoration occasioned by penetration of the rays into the tissues of the base could not be determined. At any rate the normal coloration supervened after some delay. Perhaps I ought here to say that this eyelid skin graft, of which very frequent use is made in our service for small repairs on the face, is a very reliable resource. It is rather surprising how much of the upper eyelid skin can be taken

without injury. There is never more than a temporary lagophthalmus, the motion of the lid apparently producing the necessary accommodation between the skin and the tissues below it. A point worth noting is that the graft should be taken in a fusiform pattern, with the incision lines crossing at each end. This, of course, is in the interest of the lid, since the purpose is to facilitate approximation.

4. A fourth case was that of a girl who had been burned, by fire, when quite young, with extensive injury on and under the chin. Off and on for 11 years there had been treatments by X-ray in the hope of getting rid of the contractures and the blemish. It seems an odd bit of reasoning to assume that treating a burn by another burn would effect a restoration, and in fact the result was quite the opposite. There was a continuous accentuation of the lip ectropion and the scar on and below the chin became more and more contractile. Here the repair was in two stages. After elimination of the scar below the lip, a cover was provided by advancement of the skin from below. This left a secondary defect, which was covered by a Thiersch graft. At the side of the mouth excision was followed by approximation, blending nicely into a single line. Implant of a small graft from the upper eyelid was made to complete the restoration nearest the mouth.

5. The fifth example is, if I am not mistaken, one that has been alluded to by Dr. Staige Davis in one of his articles. A physician under X-ray treatment for pruritis ani, was very badly burned, and subsequent attempts to correct one form of burn by other burning applications might well be termed disastrous. He was in a pitiable condition when he was persuaded to allow the substitution of a Thiersch graft for the much abused skin. The situation was, as may be supposed, one that presented unusual difficulties in the postoperative period, but the epidermic graft once again demonstrated its utter dependability. It relieved him of everything except his indignation, which was still vigorous when he left for home, and while I have not heard from him since, I would not be surprised if it continues to find expression.

6. A young man was referred some time ago who had an X-ray burn on the ball of his foot. The surface condition was painful, and in addition there was an interference with function obliging him to use a crutch. Dissection revealed that as a result of the burn the ligament had been enmeshed in the other tissues. Thus, it had first been immobilized and then become contractile. Dysfunction was obviously about to be definitely succeeded by distortion. However, it was still possible to free the ligament from its attachments, this patient being more fortunate in that regard than one to be men-

tioned later. After elimination of all the scarred tissue, the defect was covered by a full-thickness graft from the abdomen. Healing was uneventful. The pain has disappeared, and he now walks without crutches and experiences no inconvenience.

7. A rather striking example of the failure of radiotherapy in dealing with angioma is presented by another experience. A young woman had been given repeated treatments in the hope of eliminating a tumor which spread over the lower lip and well back into the mouth, ending only at the pterygoid region. There were areas of complete devitalization, notably on the inner surface of the lip. The tumor itself was vigorous and apparently unaffected by anything that had been done.

Separation within the mouth having revealed the extent of the tumor, the vessels were clamped above it and the tumor was dissected out. The membranes were approximated. Next the divitalized tissues on and within the lip were cleared away, and were replaced by a flap of mucous membrane from the sound side. Incidentally, this effected a substitution of the vermilion border. Both the functional and esthetic improvements in this case were rather gratifying.

8. Still another example of temporizing with angioma was that afforded by a young woman from Pennsylvania, who appeared to have a double nose. She had in fact an angiomatus condition involving both sides of the nose and extending up over the forehead and under the hair, the line of which was low down on the forehead. She had had many treatments with radium, X-ray, carbon snow, etc., and also some surgery. There was much skin destruction over the whole area. With a view to conserving as much as possible of the skin that remained good, incision was made midway of the width of the tumor, the incision line running a bit to one side of the nose. The softer tissues of the nose, but not the nasal bones, were found to be invested. On the forehead dissection revealed that the frontal bone was all gone, a fibrous layer separating the tumor from the dura. Extirpation of the tumor and correction of the nasal deformity was effected at the same operation. A large area of the devitalized skin was sacrificed and what good skin remained was brought together after extensive undermining. Incidentally, the hair line was raised to the normal position. The most fortunate part of this experience was that the heaviest burning was nearest the line that served for the incision, the most completely devitalized tissues being those that could best be dispensed with.

9. We come now to a case which, I am afraid, must serve as an illustration that there are limits to the capacity of reconstructive surgery to serve as a repair station for the accidents of radiotherapy.

The patient, a young man, whose father is a dermatologist, was treated some months ago for ringworms, one on each foot and of malignant type. The effects of the treatments seem to be out of proportion to the dosage mentioned. The ulcers are at the root of the second and third toes of the right foot, and are deep, crater-like, with indurated sides and sloughing base. The legs are red and sensitive to above the ankles, and the feet are red and mottled, with incrustations over the whole area, especially between the toes, where the sensitiveness is so great as to preclude cleansing. Both the feet are flexed inward.

The visible area was enlarged three times by the dissection. The induration was of such degree that the edges of nearly or quite 20 knives were turned in the process. The dissection was by layers, in the hope of liberating the tendons and restoring function, but these were found to be indistinguishable from the mass. Out of the whitish tissues here and there a vein would emerge and bleed smartly. The farther the dissection went the harder the scar became. No attempt was made to go beyond the tendon location. Elsewhere softer tissues were found at the base of the ulcer. An epidermic graft was placed over the dissected areas.

The solution of this problem, which is that of a radio burn complicated, no doubt, by the obscure conditions accounting for the ringworms, seems to be by surgery other than reconstructive. (Later: This apprehension has happily proved to be unfounded. The distressing conditions have all disappeared.)

10. These examples may conclude with two cases in which the effects of fire burns on the face were sought to be mitigated by radiotherapy, with consequences the opposite of those intended. The first was that of a girl who had been severely burned on the face, with much skin destruction on one side, extending to the eyelid, which was drawn up until incorporated with the brow. The resulting dysfunction had been aggravated by frequent applications of X-ray. The scar was harder in quality and harder to deal with on that account. One consequence was the failure of the first flap that was applied to the lower part of the face. Being sutured to tissues that turned out to be devitalized, it did not "take" and presently sloughed. After correction of this condition the same tubed flap was employed again, this time with success. There had been a previous repair of the lower lid, at the Roosevelt Hospital, by an epidermic graft, but the pull down of the scarred skin had reproduced the ectropion, which was corrected after the face had been dealt with. The upper lid condition, which involved some loss of the cilia, was corrected by

bringing down a line of the brow hair when the skin of the lid was reelased, and covering with a free graft taken from the sound upper eyelid of the other side.

11. The other case had some of the same essentials, the burn being very extensive and disfiguring and the induration on one cheek intensified, as in other instances, by ray treatments intended to soften the burned skin. Here also there was experience of difficulty with the flaps resulting from the presence of devitalized tissues. In the end, however, after an exhibition of great patience by the victim of the accident, a new face cover was supplied, the interesting feature of which is that three types of graft were used, a pedicle graft on the lower part, a full-thickness graft on the upper, and an eyelid skin graft for the release of contractures at the canthus. At first the coloration of the largest graft left much to be desired, but it is a satisfaction to note that it is now merging into the general color scheme of the face. In this respect, gentlemen, there is the comfort that time is always, and progressively, a dependable ally.

From particulars such as these it seems almost unnecessary to attempt to generalize. We see that mistakes are still being made, for some of the worst of the conditions presented are of very recent date. It would appear that there ought to be a revision of some of the premises on which those who have recourse to radiotherapy base their procedures. If there is any remaining belief that burnt skin can be softened by resort to another agency which burns, that belief ought to be revised. And certainly some of the conditions described suggest that there is a very imperfect conception in some quarters as to the depth to which the rays penetrate and of the effect upon tissues which lie far deeper than those layers of the skin they are desired to affect. It goes without saying that if there is recourse to reconstructive surgery necessitated by the sequelae of these therapeutic measures, the first and most essential step is the removal of those tissues that have been devitalized, and the extent and depth to which this dissection must be carried would probably surprise many of those who, in all good faith and in perfect dependence on modern methods and the latest accepted theories of the action of light and other emanations, have unwittingly produced these conditions. There is satisfaction in the knowledge that reconstructive and reparative surgery can and does find, in transference of healthy tissues, the means of correcting most of these mistakes. But that is not true in all cases, and the hope must be that, with increase of knowledge and experience, the mistakes may be avoided altogether.

636 Fifth Avenue.

AN UNUSUAL MASTOID COMPLICATION.

Dr. James A. Fisher, Asbury Park, N. J.

Loss of speech, muscular co-ordination and spastic paralysis in both upper and lower extremities. Operation, with complete recovery.

The patient, George M., age 7½ years, was carried into my office on a pillow the afternoon of Aug. 2, 1926, having been referred by Dr. C. M. Trippe, of Asbury Park, for immediate operation.

He was completely emaciated, weighing but 38 pounds, although he was 4 feet 1 inch in height. He appeared to be completely paralyzed from head to foot, unable to talk, swallow or move arms or legs. There were choreic movements of his left arm and leg, but complete flaccid paralysis of the right arm and leg. The left half of his face was completely paralyzed as a result of a previous mastoidectomy performed two years ago, and since that date, his left ear had discharged at intervals and was discharging at the time he entered my office.

On July 5, 1926, he became listless and developed a well defined chorea and was rapidly losing weight. One week previous to our examination, his family physician recommended that he be brought to the seashore. He grew steadily worse, and on July 29 he suffered an epileptiform attack and lost the power of speech and ability to swallow, the food regurgitating into his nose. He could not answer questions or carry on any conversation. His bowels had not moved for two days, but his urine was under control. He was apparently rapidly losing control, not having taken water or nourishment for several days.

Dr. Trippe observed the definite right Babinski, absence of the right epigastric and abdominal reflexes, complete paralysis of the right extremities, together with the before-mentioned pressure and irritation, cerebral symptoms, and diagnosed a temporosphenoid extradural abscess on the left side.

Rapid and pronounced emaciation are very characteristic symptoms of cerebral abscess, and when present are very characteristic, as in this case. The aphasia almost always accompanies brain abscess of the temporosphenoidal lobe when occurring in the left side

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in righthanded persons. The paralysis of the throat muscles is due to disturbance in the corticopyramidal tract, producing a typical palatal and pharyngeal muscular paralysis.

The case was operated, as stated before, on the afternoon of Aug. 2, at the Monmouth Memorial Hospital, of Long Branch, N. J. Incision was made through the postauricular scar of he previous mastoidectomy, however extending above and well forward over the temporal region. A large unhealed cavity was found posterior and above the mastoid antrum, 1 c.m. in diameter, filled with soft granulations, together with typical malodorous cholesteatomatous debris. Beneath this cavity, the floor and roof of the same was of very soft necrotic bone and entirely porous. This bone was removed and the area well enlarged, extending into healthy bone. There were no actual granulations on the dura or no free pus found in contact with the dura. There were no explorations into the dura or brain substance, as no direct pathology was detected in our exposed area. The usual radical mastoid operation was completed and the canal lightly packed with gauze tape dipped in 2 per cent mercurochrome.

For two days following the operation, the outlook seemed hopeless, but on the third day he uttered a few words and from this point on his recovery was rapid.

Without operation, these cases are practically always fatal. Complete cure rests in very early and complete operation and drainage. Even with operation, there are at times lasting signs of brain injury due to subarachnoiditis or adhesions between the dura and pia or dura and the field of operation. Thus far this case has presented no symptoms whatever of such complications.

I last saw the boy in my office on Sept. 3, 1926, walking and talking as any normal child. The mastoid cavity was completely healed. His weight had increased to 49 pounds. On Oct. 29 his mother reported continued improvement in every way.

Jersey Central Building.

FOCAL INFECTION IN THE EAR, NOSE AND THROAT, AND RENAL DISEASE.

DR. M. M. SAVAGE, Baltimore.

The subject of focal infection has apparently been thoroughly covered since Frank Billings, in 1914, published his paper on "Focal Infection".[1] At that time he justly concluded that the usual site of focal infections is in the head, and that the removal of a focus of infection is demanded as a fundamental principle in the treatment of systemic disease, especially of the chronic type. The subject of focal disease is still a favorable topic of many a medical writer in this country. In Europe, however, particularly in Germany, there are many doubting Thomases on this subject, and on my visit to Vienna, in 1921, I met a number of disbelievers; myself a believer, I have no use for the agnostic.

Infected teeth, diseased tonsils, infected nasal sinuses and mastoids constitute the *prima causa* of a large number of systemic diseases of focal infection origin. As oto-laryngologists we are chiefly interested in focal infections originating in diseased tonsils, paranasal sinuses and pathological ear conditions. It is my intention to limit myself to the renal complications. I wish to report, in brief, three cases, each typifying a kidney complication of focal infection origin of infected tonsils, sinuses and mastoid, respectively.

S. F., age 20 years, salesman, had a mild attack of tonsillitis in 1914. His urin became smoky simultaneously and showed some albumin and a few hyaline casts. From that time until 1916, when I enucleated his tonsils, he had two other attacks of tonsillitis with a repetition of the same renal complication. I am reporting this case as a *e multibus unum.*

Mrs. M. C., age 35 years, mother of three children, gave a history of antrum empyema on the right side, for which she had an intranasal operation three years ago. She was relieved from the acute symptoms, but had profuse nasal discharge. I saw her first on March 30, 1927. Ten days ago she contracted a severe head cold, with complete obstruction of the left nostril, headache and fever; a few days ago her left face became swollen. Her temperature was 101°, she complained of neuralgic pains in her head. Left side of

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face was swollen, red and tender. Left nostril completely obstructed, mucous membrane red and edematous. Routine treatment and puncture and lavage failed to relieve her. She entered the Mt. Sinai Hospital April 3. The laboratory report showed that her urine contained albumin, pus, casts, red blood cells and white cells. Suction, irrigation, etc., failed to give her relief. On April 5 I made an intranasal opening in the inferior meatus, after being compelled to remove the anterior end of the inferior turbinate. *Laboratory Report:* R. B. C., 3,360,000; W. B. C., 13,300; blood urea N. 56.0 m.gm.; phthalein test, first hour, 75 c.c., 20 per cent; second hour, 75 c.c., 20 per cent; total, 40 per cent. Urine also showed hyaline and granular casts, besides pathological constituents already mentioned. About six hours after the operation she had a chill and her temperature rose to 103° and she looked toxic. After a consultation with the chief internist I was assured that her condition was due to an acute nephritis. The antrum was irrigated daily, suction was used, and 1 per cent mercurochrome spray. The swelling of the face subsided in a few days, the constitutional symptoms also gradually improved and her urine showed barely a trace of albumin and a few hyaline casts on the day of her discharge from the hospital in April 11. The antrum suppuration did not clear up completely, necessitating three months later a Caldwel-Luc. With the closure of the intranasal opening and with stoppage of drainage she complained of headache, backache and her urine again showed albumin and other abnormal constituents which promptly cleared up after the radical operation.

The third case is one of mastoiditis with nephritic complications; Helen G., age 12 years, of Polish parents. When first seen on April 7, 1926, she was a very sick child. Her left ear was discharging for four weeks; two days ago the ear discharge stopped, with a recurrence of earache, also pain behind the ear.

Several days ago her upper and lower extremities became swollen. Her temperature was 102° and pulse 120. Both of her hands and left elbow were swollen and so were both ankles. There was a marked protrusion of the left auricle, postauricular redness and swelling, also extreme tenderness over the same area. The meatus contained a profuse yellowish discharge. She was immediately sent to the hospital to be prepared for a mastoidectomy. The preoperative laboratory report was as follows: Urine: color, yellow; reaction, acid; spec. gr., 1,018; albumin, ++; amorphous, +; casts, 0; epithelium, +; pus cells, ++; blood, ++; bacteria, +++. Blood: erythrocites, 4,480,000; leuc., 134,000; large lymphoc., small lymphoc., and large monoc.=24 per cent; polys., 76 per cent.

The diagnosis on the history sheet was: Acute mastoiditis, acute nephritis and plyelitis.

Patient was operated on on the day of her admission to the hospital. On opening of the mastoid cortex I found much pus and granulations. The anesthetic comprised of a little of each, novocain infiltration, nitrous oxid gas and ether.

April 9, two days after the operation, the history report reads: Wound doing well, no complications locally or generally. Edema of the eyelids, wrists and ankles much subsided.

April 11, urinary report: straw colored, cloudy, albumin trace, epithelium, +; pus cells, ++; blood occult.

April 16, the day of her discharge, patient feels fine, all the swellings gone. The wound had but little secretion and had a healthy look. In general, she did as well as any uncomplicated mastoid case. Urine showed but a trace of albumin and a few pus cells. The wounds closed up in three weeks, ear dry and hearing good.

In 1910, Hanau W. Loeb[2] reported cases of acute nephritis following acute tonsillitis, and stated that acute nephritis results from acute tonsillitis far oftener than is generally believed. Norton L. Wilson,[3] in 1914, reports four cases of nephritis and four cases of rheumatic fever following typical attacks of tonsillitis. In six of these cases the tonsils were removed and prompt cures followed. One nephritis case died of acute septic endocraditis. A. A. Osman,[4] in his clinical study of 56 cases of acute nephritis in children and young adults, reports that the original illness appeared to have followed some acute inflammatory process in the upper respiratory tract, usually the tonsils, middle ear, or both. This appeared to be the case in 33, or 59 per cent, of the cases. From the records of 235 cases of nephritis reported by others in the same journal, 86 cases, or 36 per cent, acute tonsillitis was given as the cause. The same author goes as far as to deny that scarlet fever is the most frequent cause of acute nephritis in children, and says, "By far the most common cause of acute nephritis in children is infection of the throat and upper air passages".

S. J. Crowe[5] reports the result of tonsillectomy in 18 cases of nephritis. Four had mitral disease and two had also had chorea. Following the tonsillectomies, in the majority of cases the urine cleared up completely.

Wichert[6] advises removing the tonsils as a routine measure in every case of chronic nephritis, unless there are special contraindications.

F. W. Törne,[7] who investigated 52 cases of nephritis in which operation was performed, is not so optimistic about the results after tonsillectomies. He says: Practically all of the patients who showed albumin and abnormal constituents before operation showed a definite increase after operation. He admits, howeved that freedom from further infection from the throat is of great advantage and may overbalance the immediate disadvantage.

I. M. Cutler[8] states: "The primary focus of infection may be in the paranasal sinuses, tonsils, teeth, etc. The most common site is in the head, the teeth and tonsils being the chief offenders." Cutler also refers to Dick,[9] who studied the bacteria of the urine and isolated from the teeth and tonsils organisms identical with those found in the urine. He showed where migration of bacteria from foci of infection takes place the bacteria are often present in the urine. All of his cases showed renal lesions. Generally the streptococcus, staphylococcus and pneumococcus have their habitat in the sinuses, teeth and tonsils and these organisms are frequently responsible for renal disease.

Systemic manifestations of nasal sinus infection is not uncommon and many writers refer to them. Among those who call our attention to nephritic complication in children are A. H. Byfield,[10] who claims that among other disorders brought into more or less association with sinus infection is nephritis and pyelitis and that there is some evidence to suggest that the chronicitiy of the renal process may be due to sinus infection.

M. F. Arbuckle[11] has seen in St. Louis Children's Hospital and in private practice well authenticated cases of cardiac disease, nephritis, arthritis, bronchitis, etc., in which the cause apparently was suppurative disease of the paranasal sinuses. He gives the clinical history of a girl, age 4 years, admitted to the above-mentioned hospital with acute parenchymatous nephritis. He found pus coming from above the middle turbinates on both sides, which on culture proved to be staphylococcus aureus. On admission the output of urine was 75 c.c., much albumin and many hyaline casts. Her eyes were almost closed by the edema; the skin over the abdomen and legs was tense and shiny. She was and had been for some time on a restricted and salt free diet. Under conservative treatment for her nasal suppuration for a week, the output of urine increased to 1,300 c.c. with the disappearance of edema and ascites.

The association of renal complications with mastoiditis has been observed by many otologists and internists. F. Adams[12] reports

three cases of acute mastoiditis with acute concurrent diseases, one of which was acute nephritis in a boy, age 14 years, who made a complete recovery 21 days after the mastoidectomy. The same author also quotes G. F. Keyper,[13] who reports two cases of mastoiditis complicated with acute nephritis, one of which recovered from both diseases. The second succumbed from the nephritis. Adams also refers to J. A. White,[14] who reported a boy, age 7 years, who developed a nephritis five weeks after mastoidectomy, with an intercurrent attack of erisypelas over the mastoid area and a furuncle over the antrum, with the healing and recovery of the otitic complications and the complete clearing up of the urine.

Volhard and Fahr[15] summarize their conclusions concerning the etiology of nephritis as follows: *1*. The pathogenic cause of every nephritis is almost without exception a bacterial infection. *2*. The different forms and stages of nephritis have the same etiology. They claim that the streptococci play the most important role in the etiology of nephritis and that the pneumococci take the second place. They say that there is no infectious disease which at one time or another does not produce a nephritis. In their table on the etiology of nephritis of 179 cases, they show that the anginas were 53 times the cause, about three-fourths of all causes; and otitis and rhinitis, 23 times. Tice[16] in commenting on Volhard and Fahr's table, says: It shows the relatively great importance of tonsillitis and conditions likely to be associated with persistent streptococcic infection, such as the influenza-like diseases, otitis media and sinus infection in the causation of the end stage of diffuse glomerulonephritis, as well as the infrequency with which the end stage occurs in cases due to scarlet fever.

Loncope and his collaborators,[17] with the assistance of Dr. Crowe, of the Oto-laryngological Staff, made a study of 40 cases of acute or subacute glomerulae nephritis treated at the John Hopkins Hospital. He reported that tonsillitis occurred in almost 53 per cent of the cases, while infections of the nasal accessory sinuses, throat and respiratory tract taken together were found in 85 per cent. Hemolytic streptococci prevailed as a cause of these infections, since they occurred in 81 per cent of the infections studied bacteriologically. All the strains of the streptococci tested produced so-called "toxic filtrates", and it seems possible that such tonxins liberated by the growth of streptococci and eliminated through the kidney might cause glomerular nephritis in patients rendered highly susceptible in some way to these toxins.

The oto-laryngologist, therefore, in his daily work has not merely a local condition to contend with, but should feel the added responsibility of either preventing or curing serious complications, if possible.

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1720 Eutaw Place.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF LARYNGOLOGY AND RHINOLOGY.

Regular Meeting, March 28, 1928.

(Continued from page 568.)

On two different days the ocular fundi were found normal. The X-ray report by Dr. F. M. Law showed moderate involvement of the frontals from granulations; the right ethmoid moderately and the left severely involved; the sphenoids showed a moderately severe involvement. The sella turcica was normal, and there was no apparent intracranial tumor pressure, so far as the Roentgenographic findings were dependable.

Operation: Dr. Wilson decided that since the patient had a definite history of sinus disease and the symptoms, the few signs, and the X-ray examination all pointed toward a lesion in the left sphenoethmoidal region, it was justifiable to perform an exploratory intranasal operation, and delegated the writer to take care of the case. On Oct. 22, 1927, under local anesthesia, the septum was resected, the middle turbinate was removed, and the ethmoidal cells were exenterated. The anterior and middle cells were found to be sclerosed, almost completely ossified, without cellular spaces. The posterior cells were contrastingly soft, necrosed and degenerated; one large posterior ethmoidal cell extended backward and was lying on top of the sphenoid; it contained thick, greenish pus encapsulated by a dark purple, soft, friable and gangrenous mucosa. The sphenoid was opened and found to be empty. The horizontal bony partition separating the two cavities was removed, converting them into one.

There was a decided improvement after the operation, and a miraculous change in the personality of the patient. She left the hospital within a week after the operation and reported regularly to the ambulatory service for aftercare. All her acute pains and aches had disappeared and she was cheerful and gained weight. Although the rapidly forming and exuberant granulations around the opening were repeatedly cauterized, the secretions had a tendency to stagnate at the bottom of the sphenoid; accordingly, on Nov. 14, under local anesthesia, the sphenoidal face was removed flush with its floor, by means of a large sized Hajek punch. Since then not a spot of granulation tissue has appeared. Up to the present, she has had no recurrence of any of the symptoms.

The case is of interest because of the severity of the symptomatology and its close resemblance to migraine, an intracranial lesion like a gumma, an abscess or neoplasmic growth, or a functional disturbance like hysteria, neuropsychosis, and even a follower of the sphenopalatine ganglion complex might have been tempted to explain the condition. Who shall say that an injection of the ganglion region might not have brought another victory, so far as relief of the pain is concerned? However, judging from this case, it is safe to state that many similar types of closed empyema are probably going the rounds unrecognized. It appears that it were better to err on the right side in performing an exploratory operation in such instances. Would it be an exaggeration to conjecture that, if this patient had not been operated upon, she stood the chance of having as a future complication an erosion of the skull plate, with a rupture of the cell contents into the cranial regions?

DR. WILSON: When I saw the case there was only a slight difference in size between the right and left middle turbinates. It was pale and had no pus at any time. The patient was under my observation for three days, and we finally came to the conclusion that there was a closed abscess in one of the ethmoid cells. It simulated almost everything you could think of intracranially.

DR. IMPERATORI: I am not making any criticism on this particular case, but there are many types of cases similar to this one that are entirely due to pressure by the middle turbinate and the mere act of removing this, or in doing a submucous resection, will give relief. I do not say that for this case, as I do not know the complete history and if the Doctor said he found pus, that certainly was a pathological condition, but I wish to direct attention to the other condition.

DR. LOBELL, closing discussion: I wish to add that at the operation, a smear was taken from the pus found in the ethmoidal cell and forwarded to the laboratory. Dr. Eggston reported that there was a Gram positive diplococcus with short chains. We have the right to say, therefore, that there was a definite bacteriologic factor in this case.

Report of a Case of Blindness in the Right Eye, Due to Retrobulbar Neuritis. Rapid Restoration of Vision Following an Intranasal Ethmoid Operation. Dr. Louis Hubert.

History: M. B., a woman, age 53 years, born in U. S., came to Dr. McCullagh's Throat Clinic on Jan. 28, 1928, complaining of blindness in the right eye. Her previous history has no bearing on her trouble, with the exception of the occurrence of frequent nasal colds. On Jan. 15, 1928, she suddenly felt a mist over her right eye, and on Jan. 21 she came to the Manhattan Eye, Ear and Throat Hospital Eye Clinic of Dr. Thompson. There she was examined by Dr. Skeel and other members of the staff. At that time the vision in the left eye was 20/30, the right eye could distinguish only hand movements. Ophthalmoscopic examination of the right eye showed a slight edema of the retina, with engorgement of its bloodvessels and a slight blurring of the disc at its margin. The left eye was normal. The nasal sinuses were X-rayed on Jan. 26. Dr. Law's report was as follows:

"The frontals are fairly large and the left shows a mild involvement from thickened membrane. The right shows a moderate involvement from granulations. Both ethmoids show a mild involvement from thickened membrane. Both antra are large and show a mild involvement from thickened membrane, especially the right. The sphenoids are clear."

Above were the history and the findings when the patient was referred to me on Jan. 28, 1928. The nasal examination did not reveal any definite pathology of nasal sinusitis. The septum was deviated to the left. The right middle turbinate was enlarged, showed some congestion of its blood vessels, and was in contact with the septum. There was no pus or mucopurulent secretion anywhere visible in the nasal cavity. She had a few bad looking teeth and her tonsils did not appear diseased. Her eye condition had become worse. She could not recognize hand movements any more with the right eye. She only had perception of light. On Jan. 31, I did a right intranasal ethmoid operation. Some of the ethmoidal cells were easily broken down and showed polypoid degeneration of the mucous membrane. Other cells, especially the posterior ethmoidal, surrounding the sphenoid, were sclerosed and very hard to open into. The sphenoid sinus was also difficult to get at and contained a mucoid secretion. Although the patient did not complain of any pain before the operation, she had severe occipital pain for a week following the operation. Within a few hours after the operation the patient volunteered the statement that it seemed to her as if a veil had been removed from her right eye. Her vision steadily improved. On Feb. 4, she could count fingers at 4 feet. On Feb. 21, her vision was 20/70, on Feb. 23, 20/50, and now her vision is 20/40. Dr. Skeel informed me that the eye ground has cleared up and appears normal.

Comment: We know that some patients suffering from retrobulbar neuritis recover without operation and without any definite diagnosis as to the cause of the trouble. We also know that many leading ophthalmologists are unwilling to accept optic nerve blindness as a result of a supposed nasal sinusitis, when such a condition cannot be demonstrated by the rhinologist. There is much disagreement as to the cause of retrobulbar neuritis and as to the kind of treatment indicated. There are most probably several types of retrobulbar

neuritis. In one type, to which the above case belongs, the optic nerve is involved by extension of inflammatory foci from the posterior ethmoidal and sphenoidal sinuses, resulting in an edema of the optic canal sheath, with venous stasis and pressure.

In a case of this sort it might be possible for the eye to recover without operation, but it might also become permanently blind. Therefore immediate operation is indicated, especially if the vision is getting worse. There is probably another type of retrobulbar neuritis, in which the optic nerve is involved by a toxin coming from a focus anywhere in the body. Leon White, of Boston, inclines now to this view. Ruttin, of Vienna, also favors the toxic theory, calling attention to the fact that in most of the cases he has seen he also found a toxic involvement of the eighth nerve. The difficulty of diagnosis and treatment is still greater increased by the occurrence of a third group of so-called benign cases of retrobulbar neuritis, similar to peripheral facial paralysis (Bell's palsy). These cases get well with or without treatment.

We should not forget that there is another type of retrobulbar neuritis, which is an early or only symptom of a multiple sclerosis. Therefore, each patient with retrobulbar neuritis needs a careful study and close co-operation by the ophthalmologist, rhinologist and neurologist.

Optic Neuritis. Dr. J. D. Whitham.

The patient was a man age 40 years. Twenty years ago he had tuberculosis of both feet and both legs were amputated at the hip. After that he was in good health until two years ago, when suddenly he had a swelling around the right eye and cheek, and became blind in the right eye. At that time some nasal operation was done, but as far as I could see when I examined him little more had been done than to remove the septum and a portion of the middle turbinate. His right eye was the same as when I saw him; he could distinguish only the fingers on the temporal side.

He came in to Dr. McCullagh's Clinic on his little wheeled platform, pushing himself along with both hands. He is a beggar in the subway. He told me that he had lost the sight of the left eye three days before. He could count only fingers 3 feet away with that eye. His Wassermann test was negative. He had an optic neuritis.

I operated and cleaned out the sinuses; the septum being almost gone, I took off the whole of the face of both sphenoids, throwing them into one cavity, and did a complete and careful nasal ethmoid (operation) on both sides. This had no effect on his vision whatever. After waiting for two or three weeks, we started injecting him with tuberculin, and when that had been given for three months his vision returned in the left eye. The right eye, however, showed no change whatever.

DISCUSSION.

DR. JASPER: I would like to ask Dr. Whitham what factor determined the giving of the tuberculin.

DR. WHITHAM: The only reason for giving the tuberculin was that he gave a history of having had tuberculous involvement of both lower extremities 20 years ago.

Resector for Removing Shaped Piece of Rib Cartilage for Nasal Transplant. Dr. C. H. Fornell.

DISCUSSION.

DR. WHITE: I hope the instrument will prove to be a success, for anything that will help in getting out the grafts will be a great aid. The hardest part of the work to me is the getting of the graft. I have been using a very small Seiler knife, and by rapidly drawing it toward me I get out a piece the size and length I wish the graft to be; and it has a bevel, and when I insert the

graft I hold the bevel side up, thus giving a more normal appearance to the bridge of the nose.

DR. WILSON: The difficulty with the chisel is that one is apt to get the graft deeper or shallower than is desired. I have not tried this instrument on the living, but on the cadaver it works very well.

DR. LORE: Dr. Fornell is to be congratulated on presenting a new instrument that apparently has a lot of merit.

Osteofibroma of Nose. Dr. John M. Loré.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. ROBINSON: Osteofibroma is not common in the nose, but is seen more often in the long bones following trauma. The reaction seen is partly due to the radium and partly to the sinusitis. The treatment consisted of the implantation of a 50 m.g. tube of radium on three occasions for 16 hours. Caustic doses of radium were used to obliterate the remaining tumor tissue, as was done in the case of carcinoma of the larynx reported in discussion this evening by Dr. Faulkner.

Cancer; Acute Perforation of Cheek. Dr. Francis W. White.

H. G., a woman, age 48 years, six weeks prior to admission noticed an ulceration on the right cheek, at the edge of the upper set of false teeth. This increased gradually until an area about 1½ inches in diameter had sloughed away. Externally, the tissues were markedly swollen, reddened and indurated. At the apex of the swelling there was a minute opening. The patient did not appear ill and complained but little of pain. The necrosis gave rise to a terribly foul odor. Up to this time she had received 11 doses of salvarsan at another institution, although the Wassermann reaction was negative.

On Jan. 18, 1928, the patient was admitted to the Manhattan Eye, Ear and Throat Hospital, and laboratory tests were instituted. Smears and scrapings from the ulcerated area showed prolific growth of Gram positive and negative organisms, Vincent's streptococci in short chains, nonhemolytic pyogenic cocci, many mycelial forms of Gram positive organisms with clubbed ends, probably of the actinomycosis group.

Ascending doses of potassium iodid were given, but glandular swellings about the neck developed, which gradually subsided when the iodids were discontinued. The foul odor from the necrotic area gradually disappeared, and the cavity became clean and showed many granulations.

On Feb. 9, the patient developed erysipelas of the right side of the face, which extended to the neck of the same side, and over the bridge of the nose to the left side. Erysipelas antitoxin, 10 c.c., was administered, and this dose was repeated within 24 hours. The patient made an exceedingly rapid recovery from the attack, the temperature being normal within 14 hours after the last injection. From this time on, however, there was a marked increase in the destruction of the right cheek, and the microscopic examination of the tissues taken from the margin of the ulcer showed white and red cells, bacteria and mycelial bacteria, and cellular elements.

Diagnosis: Epithelioma, basal cell type.

Dr. Robinson applied radium as follows: March 13, 1928, 3,800 m.g. hours; March 16, 2,400 m.g. hours.

On March 24, erysipelas was apparently developing again, but an injection of 10 c.c. of erysipelas antitoxin seemed to ward it off.

Vincent's Angina After Tonsillectomy. Dr. Francis W. White.

David G., age 18 years, had a tonsillectomy performed on Nov. 10, 1927, under local anesthesia. One week later he returned, and was admitted to the

ward with a temperature of 101°, which rapidly rose to 104°, at which time I was asked to see hm. He complained of a severe sore throat, especially on deglutition of fluids. He was able to only slightly open the mouth.

The cervical glands were markedly swollen, especially on the right side. The lobe of the right ear and point of right shoulder appeared to be on a continuous line. The tissues were decided brawny, and pressure caused severe pain. The jaws were separated with difficulty and it was then seen that the entire fauces were involved in a severe inflammatory process. The pendulum uvulae was enormously swollen and the pillars were markedly involved; the tonsillar fossa of the right side was inordinately deep. A grayish-white membrane covered a considerable surface of the inflamed tissues.

Blood fixation tests were negative. Red blood cells, 4,000,000; white blood cells, 25,000; polymorphonuclears, 88 per cent; small lymphocytes, 12 per cent.

Bacteriologically, streptococcus viridans, staphylococci and Vincent's organisms were found.

The treatment consisted of irrigation of throat with warm saline solution every two hours; mercurochrome solution, 1 per cent, three times a day; one full dose of salvarsan intramuscularly, and the local application of 0.3 grams of salvarsan in half an ounce of glycerin two or three times a day.

Patient was discharged one week after admission.

DISCUSSION.

DR. HELLER: I would like to know whether the condition of the teeth was noted in the case of Vincent's angina.

DR. WHITE: Yes, there was an involvement of the gums.

DR. HELLER: I recall a case I saw some 25 years ago, before we had the Wassermann test and salvarsan, a man, age 45 years, who gave a definite history of syphilis and had mercury and iodid treatment. He had a gumma on the side of the cheek; there was no evidence of trauma. We treated him according to the classical methods, but the gumma broke down and he finally died of carcinoma.

DR. IMPERATORI: How long was it before a biopsy was done?

DR. LORE: I have always believed that those miraculous so-called cures with salvarsan were very doubtful. I recently had a very interesting experience with a man under treatment for syphilis with a lesion in his throat, and yet while under this active treatment he developed a case of angina. Dr. Jones has also had an experience with syphilis of the tonsil in which Vincent's organism was found.

DR. WHITE: As to salvarsan in Vincent's angina, I am sure that the mercurochrome and saline did not cure this man; I do think the salvarsan had some effect.

Replying to the question concerning the biopsy, after we found that the ulceration was increasing rapidly, a biopsy was done. Up to that time the condition was regarded as being due to an organism. The Wassermann test was negative.

DR. IMPERATORI: I do not wish to harp upon the question of biopsy, but this is a good opportunity to do so. If a biopsy had been done earlier in this case it could have been proved or disproved that it was actinomycosis or carcinoma, and that knowledge might have helped in the matter of the treatment. It seems that in the minds of many today, a biopsy means the stirring up of carcinomatous tissue. I do not think it does. If a patient with carcinoma of the breast has the breast massaged, etc., it has been proven that within 12 or 14 hours there will be carcinoma cells in the lymphatics of the axillae; it has also been proved that massage of carcinoma will spread it. As usually done, a biopsy should in no way greatly traumatize the tissues, and in my experience no metastasis, insofar as known, has been caused by this resection of a small piece of tissue. It has materially aided us in the early diagnosis of these cases.

DR. JONES: We have been trying out sodium biborate and have gotten very good results in cases of Vincent's angina.

Secondary Syphilis of the Throat. Dr. R. L. H. Murphy.

The first case is that of a woman, age 24 years, with six children. The other case we had was a young man who came in this afternoon with ulcerations on both tonsils; no history of any primary sore.

DISCUSSION.

DR. GLUSHAK: Referring to the first case of syphilis of the throat, I would like to know the experience of the men about the advisability of operating on the throat if there is an active syphilitic condition in the body. One is inclined to keep hands off. This reminds me of the case of a young woman who visited one of the local clinics, where a tonsillectomy was done. She went back to the clinic several time, and subsequently developed a nasal twang to her voice. When she came to me half the left soft palate was eaten away. I called up her physician and asked him if he had any reason to suspect syphilis, to which he replied in the negative. I did, and ordered a Wassermann, which showed 4 plus.

Of course it is possible that there were no signs of syphilis in the throat, but it is also possible that there might have been patches here and there which would have thrown suspicion on the conditions and have delayed operation. I should certainly think of active syphilis at the slightest provocation before operating on the nose or throat.

DR. FAULKNER: I recall a statement of my own in regard to this case. We saw it just three weeks ago; the lesion appeared on the anterior pillar and the base of the tongue, looking like a small ulcerated excrescence, with a little white slough on it. I made a tentative diagnosis of Vincent's angina. Dr. Jasper made smears, and it was negative. Then it was sent back with instructions to keep on making smears. They took four other smears and never got a Vincent.

About a year ago a doctor came to my office in great distress. He had seen two well known nose and throat specialists for a lesion almost exactly like that; he said there was also a little suggestion on the other side like an enlarged lingual adenoid. If one had had a photograph of his case and compared it with this one, I think you could not distinguish the two. This doctor also saw a general surgeon, who advised immediate operation; and all agreed that it was an epithelioma. I sent him to the laboratory and had two or three smears made before we got a Vincent; which was what it was. This case looked exactly like the other, but it turned out to be epithelioma.

DR. WHITHAM: Suggestions as to treatment were desired; I would not pose as authority in a case of this case, but I do think there is but one way of treating such condition. I think that radium would probably heal it up within a few weeks, but I am sure that it would recur, and the recurrence would be rapidly fatal. I think if a wide destruction of part of the tongue and pillar was done with the endothermy knife it would give the man the best chance he could have.

Vincent's Angina. Dr. J. G. Morrissey.

The patient was a house worker, age 50 years, whose chief complaint on coming to the hospital was a sensation as of a needle sticking in the right side of the throat; this had been troubling her for three weeks.

She had a mastoid 15 years ago, following which her tonsils were burned out. After this, she had diphtheria. Since then she had been a chronic asthmatic, having severe attacks, the worst of which occur about 4 o'clock every morning and last for two hours. For relief, she had an asthmatic powder. She suffered constantly with constipation and severe headaches, and had noises in the right side of the head, which she attributed to the right ear. Both antra were opened two years ago. The blood Wassermann examination was negative, as was also the urine examination.

X-ray examination showed the frontals to be small, with thickened membrane; the ethmoids were free from exudate; the left antrum was small. The floor of the nose showed a severe involvement from dense granulations or pus; the right antrum showed moderate involvement; the sphenoids were mildly involved. The X-ray diagnosis was, "Chronic purulent processes involving antra".

Physical examination showed deviated septum, pharyngitis, diseased tonsils. The right tonsil showed a slight discharge of pus. The eyes were apparently normal. There was a swelling of the lower jaw and neck, but no signs of inflammation.

A smear from the right tonsil, taken a week ago, showed her to have a positive Vincent's.

The treatment of the case has been irrigation and application of sodium perborate every three hours, resulting in very decided improvement.

Here is another case of Vincent's angina, treated both locally and by salvarsan intravenously, in which the result is ascribed to the salvarsan. I question that conclusion. We must not lose sight of the fact that (1) Vincent's angina in adults may and very often is associated with syphilis, and (2) that the organisms of Vincent's angina are anerobes. In a straight case of Vincent's angina any form of local treatment which will remove the debris from the ulcerated area and expose it to air will produce a cure, be it plain water, peroxid or sodium perborate. The latter two are the logical ones to use because they liberate free oxygen.

Chronic Ethmoid Treated With Radium. Dr. E. C. Kern.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

Syphilis of the Soft Palate. Dr. V. G. Smith.

The patient, M. D., had been married for six years and had two children living and well, age 2 and 4 years, respectively. Nine months ago she had an abortion at 6½ months. She gave a history of having had treatment with blue ointment at the age of 5 years, continuing for several years, apparently for congenital syphilis. There was no history of a primary sore at any time. Her father had always had throat trouble, and died suddenly at the age of 33 years. Her mother was living and well. She had no brothers nor sisters.

Her illness began on Feb. 1, 1928, as a sore throat. She came to the clinic on Feb. 6, with a large sloughing ulcer of the soft palate at the left of the uvula. The Wassermann reaction was 4 plus.

She was given salvarsan on Feb. 6 and Feb. 9, and weekly thereafter; also mercury inoculations and potassium iodid. The ulcer was completely healed on March 2, leaving only the original loss of tissue. There was practically no scar tissue formation and no loss of function in speaking or swallowing.